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CLINICAL FORMS OF LABYRINTHITIS.*

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The tympanogenous route of infection in labyrinthitis is far more common to the otologist than the meninges. There are four distinct routes of infection (Ruttin,¹ Dean and Wolff,² Turner and Fraser,³ Seydell,⁴ MacKenzie,⁵ Zange Johannes,⁶ Jansen⁷) :

1. Fistula through the round window. This is the most common.
2. Fistula through the oval window.
3. Fistula through the bony capsule of the labyrinth.
4. Through the vascular channels.

Ruttin¹ observed in his statistics that the oval window was involved in the labyrinth more often than the round window. That the round window would be involved more often is anatomically explained by Dean and Wolff.² The round window is deeply placed in the niche, the "fossula fenestrae cochleae," within the overhanging rim of the promontory. Once infection gets started in the middle ear, a mass of pus, collecting in the niche, drains from it with difficulty; whereas, the niche of the oval window, the fossula fenestrae vestibuli, is in a more exposed position than that of the round window. Paracentesis drains it more easily.

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Fistula through the bony capsule of the labyrinth occurs chiefly in the prominence of the lateral canal towards its anterior part (Turner and Fraser,² MacKenzie³). The erosion may be superficial or may involve the entire thickness of the bone.

Bacterial toxins may find their way through erosion and produce circumscribed labyrinthitis of serous type. The fistula usually is formed by the purulent secretion in the middle ear, causing a chronic necrosing osteitis, or by a cholesteatomatous mass, which by pressure gradually invades the capsule of the labyrinth.

The vascular communication between the mucous membrane of the middle ear and endosteum of the labyrinth is not uncommon route of infection (Zange¹²).

Hinsberg¹⁰ states that infection of the labyrinth is a more frequent complication of purulent otitis media than all of the intracranial complications combined. He further states that a great number of intracranial complications following a purulent otitis media is usually secondary to a labyrinthitis. Infection of the labyrinth through the fistula of horizontal canal offers a better prognosis than path of infection through the oval or round window, or even through a necrotic defect in the promontory.

Uffenorde,¹¹ in 1910, found that when the toxins enter the labyrinth it usually produces a serous labyrinthitis; whereas, bacteria which are found in the latter stage of acute purulent otitis media usually produce purulent labyrinthitis. The invasion of the labyrinth during this acute stage through the round or oval window may be bacteria, or may be due to a dialysis of toxins through the intact membranes.

In about every 100 cases of middle ear infection there develops a labyrinthitis. Logan and Turner⁴ give 1.4 per cent, whereas Uffenorde's 3 per cent is generally assumed too high. In Ruttin's 96 cases of labyrinthitis, 81 occurred in chronic purulent otitis media, ten occurred in subacute purulent otitis media, and five in acute purulent otitis media. In the latter five cases all were diffuse manifest purulent labyrinthitis. Cholesteatoma occurred in 31 cases. Tubercular otitis media is not uncommon cause of labyrinthitis. Goerke cites five examples from a series of 16 cases. Polypi are commonly

found associated with labyrinthitis, not directly causing the complication, but indirectly by its obstruction giving poor drainage and ventilation. In these cases it is always best to do a complete labyrinth examination before using the snare.

Unfortunately, the diversities of classifications of labyrinthitis propounded by many eminent otologists have shrouded this interesting and rather simple subject with a veil of mystery. It is not the writer's intention to add another burden upon an overladen and overtaxed subject, but will attempt to remove this unnecessary confusion by interpreting and evaluating the salient features of Ruttin's²⁰ classification.

1. Chronic circumscribed labyrinthitis.
2. a. Diffuse manifest serous labyrinthitis. b. Diffuse manifest purulent labyrinthitis.
3. Diffuse serous induced labyrinthitis.
4. Latent purulent labyrinthitis.

This simple and yet practical classification befits any labyrinthine picture complicating an acute or chronic middle ear infection. It is quite difficult to present any innovation in this voluminous field, but a better understanding of certain clinical symptoms and findings is a decided aid in the pursuit of uniformity.

1. *Chronic Circumscribed Labyrinthitis*: Alexander,¹³ Neumann¹⁴ and Wittmaack.¹⁵ These men have done much research in the pathology of this form. It is caused:

- a. By pressure atrophy or ulceration of the bone due to accumulation of cholesteatoma.
- b. By purulent destruction or necrosis of mucous membrane overlying canal prominence with absorption of bony wall by rarefying osteitis. Seen in acute otitis and acute exacerbations of chronic otitis. The erosion continues and absorptive process goes on until the endosteum of canal is reached and have fistula symptom. Diffuse serous and purulent labyrinthitis may result when there is acute exacerbation of chronic ear.

In cholesteatoma the process is milder and growth is slow, so that nature has a better opportunity to wall itself off by a barrier of connective tissue. However, should the aseptic

cholesteatoma become septic, and in the presence of a fistula there can readily occur a diffuse serous or purulent manifest labyrinthitis.

In the purulent destruction of the canal wall it is possible for the bacterial toxins in the middle ear cavity to find their way through the erosion into the inner ear and produce a circumscribed labyrinthitis of a serous or serofibrinous type. The majority of these cases heal up without reaching the purulent stage of inflammation.

The horizontal canal is the one usually involved. Pus may invade either the cochlear or vestibular portion of the labyrinth. There have not been any cases reported where there has been a circumscribed process in the cochlea, because it tends to rapid infection of entire labyrinth. On the other hand, one can have a localized suppuration of canal without any involvement of the cochlea.

The commonest location for fistula: 1. In the horizontal canal; 2. oval window; 3. promontory (Rae¹⁷).

Symptoms: Acute stage is similar to manifest diffuse labyrinthitis, but differential point is that the hearing is retained in the former. The attack lasts for about a week and then passes into:

Chronic stage. This is the stage at which we usually see the patient. He gives a history of intermittent attacks of vertigo. Some can describe their sensations of dizziness and others cannot. Sudden violent movements of the head or possibly bending over may excite a vertiginous attack. The uncommon cases accidentally found on examination may complain of no vertigo. Their attacks of vertigo are usually accompanied by a low grade spontaneous nystagmus. It may be absent, and when present may be to either side of the lesion. Functional examination of cochlea reveals a conductive lesion, and the rotational test on the uninvolved ear is normal while the affected ear usually shows a diminished nystagmus. The caloric test is practically always negative. We must remember in doing the caloric test with the head in the erect position, the anterior vertical canals are stimulated, and not the horizontal canals. In chronic circumscribed labyrinthitis the anterior vertical canals are rarely involved. I have observed two cases where, doing a Barany test in these

chronic circumscribed cases, marked vestibular symptoms appeared and patient developed an acute attack. If the cochlea is not involved, one cannot have intracranial complications. The semicircular canals have no direct communication with the meninges.

It is in these groups of cases that we test for a fistula. A negative fistula does not mean anything. Clinically, one can have a positive fistula test one day and negative another. This is easily explained by the fact that any obstruction, such as granulation tissue, purulent secretions, cholesteatomatous masses may occlude the fistulous opening and thereby give a negative response.

In testing for a fistula, the late Duel¹⁶ emphatically stressed the point that when once administered, do not try it again and again, for there is a possibility with forceful compression and aspiration of breaking down the delicate barrier nature is trying to protect from the meninges. A fistula test is only positive when the necrosis has extended to the membranous labyrinth but not invaded the endolymph. It is not surprising to see these cases go on for years without any diffuse attacks when we realize that the endosteum of the labyrinth has enough resistance to prevent the penetration of infectious micro-organisms into the interior of the labyrinth.

With each attack of vertigo, one will notice upon examination there is always a gradual diminution in the response of both cochlea and semicircular canals. This may continue for years until you have this form of labyrinthitis eventuate into one of three phases:

- a. This circumscribed process may be arrested with complete healing with connective tissue and then bone, with all the normal functions of cochlea and canals intact.
- b. With constant irritation either from the purulent secretions, or from cholesteatomatous mass, etc., the destruction continues until you have a circumscribed lesion of the membranous labyrinth. This form gives repeated attacks of vertigo, forcing the patient to seek relief.
- c. Destruction of lesion still continues, or the virulence of the organism in an acute exacerbation of a chronic ear stirs up a diffuse manifest purulent labyrinthitis.

2. Diffuse Manifest Labyrinthitis: a. Serous type; b. Purulent type.

There is much dispute regarding the serous form. Alexander,¹⁸ Neumann,¹⁴ Goerke,¹⁹ Ruttin,¹ Barany describe this form as a distinct entity, clinically and pathologically. It is an acute nonpurulent inflammation of the labyrinth. There is a hyperemia and a serous exudation involving the membranous labyrinth. According to Turner and Fraser,² there is an increase in the albumin of the labyrinth fluid due to increased secretion from the dilated vessels of the stria vascularis. In the microscopic secretions masses of curdled lymph or fibrin threads are observed in the peri- and endolymphatic spaces.

Clinically this serous form cannot be differentiated from the purulent form in the early stages of acute attack. The vestibular picture, however, begins to subside in the former within a few days in the vast majority of cases; whereas, in the latter, the cochlea and vestibular reactions are completely gone.

According to Alexander, the average case of suppurative labyrinthitis is probably preceded by serofibrinous form, and is due to the irritative action of the bacterial toxins generated in the middle ear that seep through the windows into the inner ear. With increased virulence the infecting micro-organisms break through the natural barriers and find their way into the inner ear when the serous form of labyrinthitis goes over to the suppurative form. After suppurative labyrinthitis is established, the soluble toxins find their way into the adjacent cavities of the inner ear after the same manner as the bacterial toxins found from the middle ear to the inner ear, and if resistance is poor, there is danger of intracranial complication.

According to Goerke,¹⁹ after suppurative labyrinthitis has set in, there later results a destruction of the nervous elements, Corti's organ, maculae and cristae with their nerve fibres, and later the more resistant parts of the membranous labyrinth. Later, granulation tissue forms along the vessels, which invade the purulent exudate from the endosteum, and raises a barrier against the spread of infection. In very rapid and virulent infections this protective layer is not formed

and meningitis results. In the last stage new connective tissue and bone are deposited. Atrophy of the VIIth nerve is sometimes observed, and is due to destruction of the spiral ganglion.

The clinical picture is typical. The nystagmus is horizontal rotary coming from all the three canals and 3° . During the first few hours the nystagmus is on the same side of the lesion due to an irritated labyrinth. Later the nystagmus goes to the sound ear, which means the labyrinth has become destroyed. Patient acutely ill, holds on to the bed, and his eyes are closed, or if opened, looks in the direction of the slow component. The vertigo is a rotary type and corresponds to the same plane of nystagmus. Patient has the impression that surrounding objects are rotating about him. Some can describe their sensations and others cannot. He lies on his sound ear ("Zwangslage"). There is usually nausea and vomiting present. Temperature is not a sign of suppurative labyrinth. Usually there is no temperature. If temperature is present, it is not over 101° and due to the concomitant lesions, *viz.*, acute otitis or acute mastoid, etc. Headache also is not a symptom of labyrinthitis but may be present.

Leave patient alone. Do not do any functional tests. May spread the infection to the meninges. Suppurative labyrinthitis *per se* is not a fatal malady, and if the patient dies, it is due to some intracranial complication.

The second day the patient feels a little better and then can do a caloric and cochlear test. If both are absent, then watch carefully. If the lesion is a serous type, the hearing and vestibular irritation will reappear in three or four days. Barany waits eight days, and if the responses do not appear, calls it a purulent type. However, this is not a strict and fast rule. Page²⁰ reports a case where it has taken several weeks or more before he was able to elicit any response.

If the lesion is purulent, the hearing and vestibular reactions are completely absent. In six weeks the patient feels fine and wants to get up. It is best, if you can, to keep the patient in bed three months. The internal ear, uncomplicated about the tenth week after the attack, begins to fill with granulation tissue, and this is called labyrinthitis granulosa. This means, though the labyrinth is in a latent form, it is

still viable, and any operation on the ear may stir this again into acute form, with a possible meningitis. This is followed by labyrinthitis fibrosa. Later this calcifies, finally resulting in labyrinthitis ossificans. This formation takes place anywhere from six months to several years (Manasse,²¹ Ruttin,²² and Wittmaack²³). This process is very fortunate for the patient, and occurs in about 50 per cent of all cases of purulent labyrinthitis.

A labyrinthitis coming on during the first 12 days of acute purulent otitis media usually is a serous type; whereas, coming on the third week, or later in the acute stage, is usually a purulent type.

The following schema condenses in a very practical way the picture of diffuse manifest purulent labyrinthitis:

Symptoms.	Acute Stage.	One Week Later.	Six Weeks Later
1. Nystagmus	↗ 3° opposite	↗ 1°	Absent
2. Dizziness	Marked	Very little	Absent
3. Nausea	Marked	Absent	Absent
4. Vomiting	Marked	Absent	Absent
5. Falling	Same side lesion	?	Absent
6. Past Pointing	Same side lesion	?	Absent
7. Hearing	Totally absent	Gone	Gone
8. Vestibular	Totally absent	Gone	Gone

TERMINATION OF DIFFUSE MANIFEST LABYRINTHITIS.

A. Serous Type: The outcome will be a normal labyrinth.

B. Purulent Type: *a.* May eventuate to the latent purulent labyrinthitis, in which the labyrinth is completely ossified and all the functions gone, or *b.* may eventuate to some intracranial complication, the most important being meningitis and cerebellar abscess.

Temperature and headache are not symptoms of labyrinthitis *per se*. The temperature as stated, is present, is usually 101°, and due to concomitant lesions, *viz.*, acute ear or mastoid, etc. However, a sudden rise of temperature without a definite reason may mean a beginning meningitis.

Headache plays a very important role as a very early symptom of meningitis. If the headache comes on early in the acute stage of manifest labyrinthitis and the purulent labyrinthitis progresses rather favorably, then this symptom may be disregarded. But, if later, this symptom becomes severe, and the condition of the patient not progressing favorably, then it is an early sign of intracranial complication.

Kopetzky²⁴ holds that the absence or diminution of the copper reducing substance in the spinal fluid is probably the earliest sign of beginning meningitis.

3. Diffuse Serous Induced Labyrinthitis: This has been first described by Alexander. It is a nonpurulent (serous) inflammation which goes directly through the walls of the labyrinth. It differentiates itself immediately from diffuse manifest serous form. 1. The latter usually complicates a chronic circumscribed labyrinthitis. 2. It has the prodromal vestibular symptoms. 3. The fistula test is always present, whereas, in the former, a. it has no relation to a chronic circumscribed labyrinthitis; b. there are no prodromal vestibular symptoms, the onset being sudden; c. the fistula test is always negative.

Diffuse serous induced labyrinthitis usually follows: 1. Acute purulent otitis media. Voss has described a series of such cases that appear in the early stages of the disease, and the onset is ushered in like diffuse manifest labyrinthitis. The differentiation is easily ascertained. In the former there is always some remnant of hearing or vestibular irritability present. However, there are occasional cases reported where the cochlea and vestibular functions are lost. In these cases, after two or three days, their functions slowly reappeared. It is possible, too, as reported by Ruttin,¹ that these cases may pass into the diffuse manifest purulent form. 2. a. In cases of unhealed radical operation, and b. a short time after a radical operation. Ruttin presents 11 such cases in his series. The induced serous labyrinthitis following a radical mastoidectomy usually appears in from one to three days. The symptoms appear suddenly, just as the patient begins to feel a little better. A traumatic injury during a radical operation manifests itself during the operation (giving a marked slow component of the nystagmus, the rapid component being in abeyance in narcosis) and immediately afterwards with its marked vestibular syndrome.

4. *Latent Purulent Labyrinthitis*: Patient gives a history of having an attack of diffuse manifest purulent labyrinthitis some time ago, and at the present time feels fine and presents no symptoms except perhaps a chronic running ear with deafness.

Functional tests show complete obliteration of the labyrinth (total deafness and no caloric reaction), no fistula symptom demonstrable. The turning test is very important in these cases, and proper interpretation of its findings are highly significant. If the sound ear gives a nystagmus 15-20 seconds on rotation, and the diseased ear five seconds' duration, it means that the labyrinth is still functioning and not totally destroyed. When ossification has set in, the sound labyrinth takes a compensatory function controlled by Deiter's nucleus, so that now, with the rotation test, either direction, the duration of the nystagmus becomes more or less equal on both sides. This compensation is known as the "compensatory phenomenon of Ruttin."

This compensatory phenomenon is untenable by many otologists; yet, on the other hand, the phenomenon has been correctly demonstrated on many cases (Neumann, Alexander, MacKenzie, Turner, and Fraser and Fremel). The importance of this phenomenon will be seen in the surgical indication for latent purulent labyrinthitis.

The following chart, according to Ruttin in personal notes, condenses in a very comprehensible fashion this entire subject of labyrinthitis:

Labyrinthitis	Previous History	Present Symptoms	Nystagmus	Hearing	Caloric	Turning	Fistula Symptom
Chronic circumscribed labyrinthitis	Vestibular symptoms in attacks.	Vestibular symptoms + or —	↔ or ← negative	+	+	+	+
Diffuse manifest serous labyrinthitis	Vestibular symptoms in attacks.	+	→	— or +	+	+	+
Diffuse serous induced labyrinthitis	Negative.	+	→	— or +	+	+	—
Diffuse-manifest purulent labyrinthitis	Severe; 3-14 days.	+	→	—	—	—	—
Latent purulent laby.	Severe; 3-14 days.	—	—	—	—	—	—

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BONE CONDUCTION IN THE EXPERIENCED PILOT AND A PROBABLE INTERPRETATION.

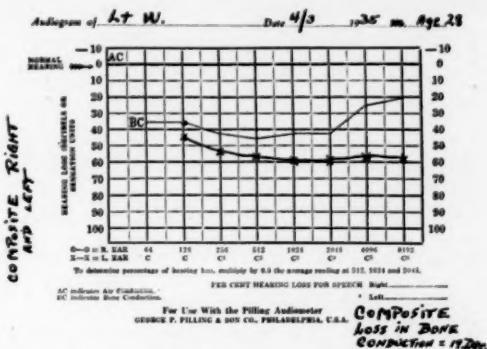
DR. CHARLES FIRESTONE, Seattle.

Aviation is a comparatively new industry. With its inception was born a host of problems to physicians interested in aviation medicine. Early in the course of aviation the physicians dealing with the selection of personnel for piloting the planes were plagued with problems of choosing persons who were optimum types from the neuropsychic personality standpoint, and from the standpoint of their abilities to physiologically and psychologically adjust themselves to the changes occasioned by high altitudes and their attendant disturbances of the metabolic processes, and from the standpoint of their aptitude as judged from stimulus-reaction-time tests. The profession itself was doing its utmost, in trial and error fashion, to determine just what class of individuals represented the optimum type for flying, and it was not until profound study and not a few grim experiences that physical and psychological standards were formulated by which the aspiring professional pilot of today is appraised. But this is not the end; for at this writing governmental agencies are at work studying the pilot in an effort to make aviation safer for the public. So much for that.

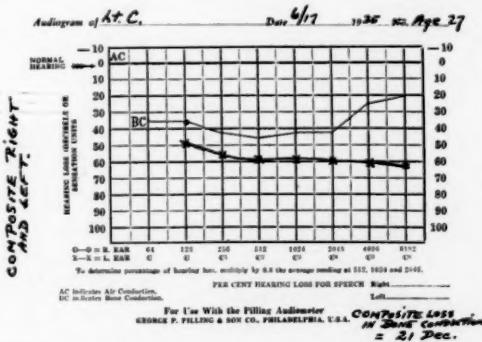
And now that aviation has reached its adolescent stage, pilots who have had from 10 to 20 years' concentrated flying experience present themselves for observation and bring with them opportunities to observe what, if any effects, prolonged flying has produced on them. In this discussion the author wishes to confine himself to an examination of the perceptive portion of the auditory mechanism of the experienced pilot. Armstrong and Heim¹ have aptly described the effects of prolonged aviation on the conductive portion of the auditory mechanism in a recent issue of the *Journal A. M. A.*

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This writer has recorded audiograms on 109 pilots during the past two and one-half years. Only pilots who have had seven to 20 years' flying experience and a minimum of 800 hours' flying experience are included in this group. The air-conduction graphs are omitted, and the bone-conduction

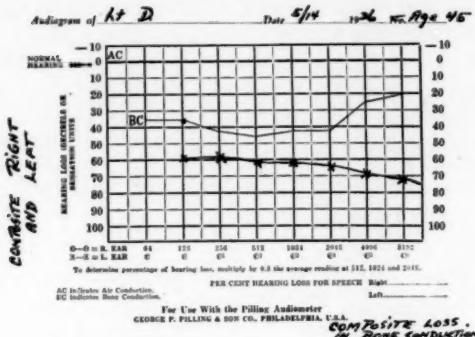


Audiogram 1. Lieutenant W., age 28 years. Flying time, 1,089 hours. Composite loss in bone conduction in both ears, 19 decb.

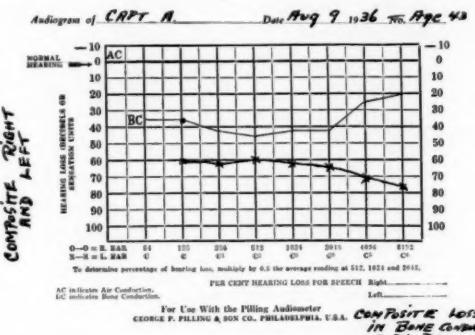


Audiogram 2. Lieutenant C., age 27 years. Flying time, 1,673 hours. Composite loss in bone conduction in both ears, 21 decb.

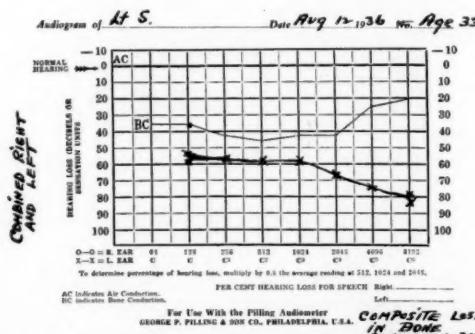
graphs are plotted as composites of both right and left ears, inasmuch as the findings in this group were such that left practically superimposed the graph of the right ear and vv. The following seven audiograms here reproduced were taken at random from the group of the 109 that were studied and are representative of them:



Audiogram 3. Lieutenant D., age 45 years. Flying time, 3,000 hours. Composite loss in bone conduction in both ears, 32 deb.

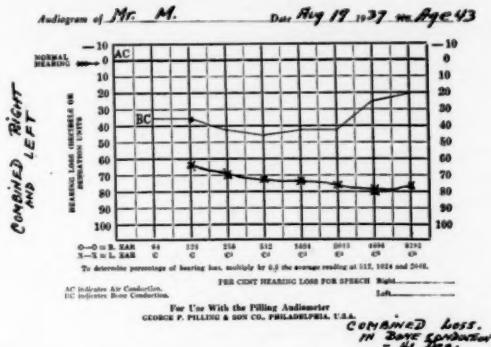


Audiogram 4. Captain A., age 43 years. Flying time, 8,700 hours. Composite loss in bone conduction in both ears, 34 deb.

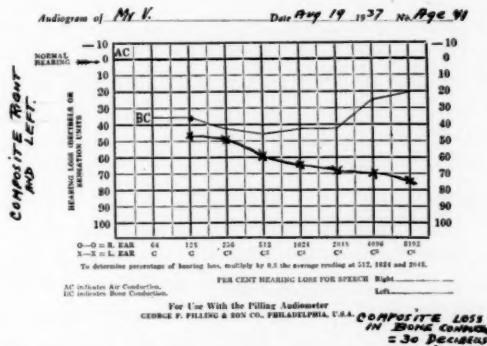


Audiogram 5. Lieutenant S., age 33 years. Flying time, 6,500 hours. Composite loss in bone conduction in both ears, 34 deb.

If one were to view these audiograms in a detached and random manner they would suggest the audiograms of otosclerosis. Let us now consider the pathogenesis by which these pilots come to yield these audiograms.



Audiogram 6. Mr. M., age 43 years. Flying time, 12,300 hours. Combined loss in bone conduction in both ears, 41 decb.



Audiogram 7. Mr. V., age 41 years. Flying time, 13,379 hours. Composite loss in bone conduction in both ears, 30 decb.

The aviator, in flying his craft, is exposed to both vibration and noise. While the harmful effects of noise on the auditory mechanism have been studied, the harmful effects of vibrations directly transmitted to the individual, that is, vibrations in which the individual vibrates in unison with machinery surroundings over a prolonged period of time, have

received comparatively scant attention if one is to judge from a search of the literature on the subject. It is true that we cannot have noise without vibration, but in the case of the aviator, the vibrations are not only transmitted to him by way of the atmosphere, but they are also transmitted to him directly through his musculo-skeletal system.

In his search for information bearing on the harmful effects of vibrations on the human organism, this writer was able to find but one discussion on the subject. F. Koelsch,² writing in the September, 1935, issue of *Jahreskurse fur Arztliche Fortbildung*, states that a study to determine the effects of vibration and noise on workers employed in German shoe manufacturing industries and in industries employing compressed air drills, revealed the following findings, namely, that workers in these industries suffer from angioneurosis of the extremities close to the points of trauma, blanching, numbness, palsy, and in general a neuromuscular asthenia. He states that these workers become so that they need increased threshold of stimuli in order to evoke responses in them. They get what he refers to as a vasoneurotic diathesis. He states that the actual pathological changes are productive changes in the region of the capsules and tendons of the joints involved. In some individuals he has observed the formation of actual exostoses. He also finds joint crepitus and radiographic changes in the affected joints. He records a diminution of hearing in all these cases, but does not state which portion of the auditory mechanism is affected.

Let us now consider the aviator whose acoustic nerves are constantly being doubly traumatized by noise of the motors and by violent vibrations of his entire musculo-skeletal system. If we consider the osseous, articular, and circulatory changes reported by Koelsch, and they are in keeping with the rationale of the effects of trauma on the osseous system in general, we may expect similar changes in the aviators' bony capsules as a result of the vibrations to which he is exposed, perhaps some productive changes in the oval window regions, coupled with a certain amount of trauma to the terminal auditory fibres produced by the noise of the motors. In effect, what we may expect in these aviators are pathologic changes akin to otosclerotic changes in the auditory mechanism. In view of this, the accompanying audiograms are to be expected.

All the pilots included in this study showed a deterioration in bone conduction. It is interesting to note that without exception the pilot, when apprised of his condition, admits that his hearing is somewhat impaired, but adds, rather boastfully, that he can detect the slightest motor noise or other motor defalcations while in flight long before his newer and less experienced co-pilot can hear them at all. Shades of paracusis Willisi!

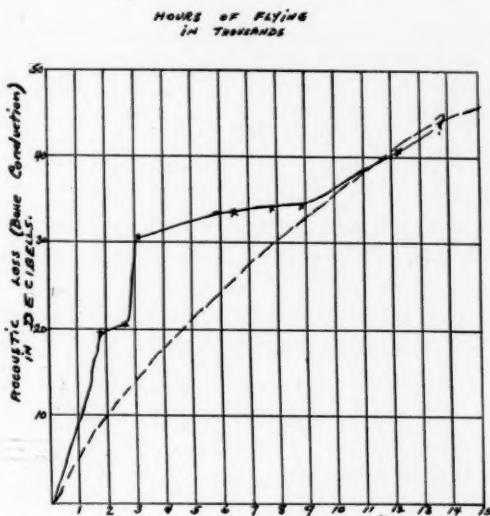


Fig. 8.

The group of pilots included in this study have had from 800 to 14,000 hours of flying experience. This writer is advised by competent authority that there are very few pilots, if any, in the world today who have had more than 14,000 hours of flying experience, and that probably no more than five or ten have had as much. The audiograms above are random and wholly representative of the group. When from this series we chart the diminution in bone conduction as related to the number of hours of flying experience, we obtain the above graph:

A cursory analysis of this graph reveals that the maximum damage suffered by the pilot occurs during his first 3,000

hours of flying, and the damage following that, while it is progressive, is markedly slowed down. The tendency of the damage curve is represented by the dash curve. This state of affairs, coupled with the aero-otitis described by Armstrong³ promises to become an entity that will confront the otologist more and more as aviation progresses, and pilots with a large number of flying hours to their credit become more numerous. Indeed, a new otologic entity seems to have arrived, that of aero-otosclerosis.

A question of preventive otology also presents itself. Can anything be done to prevent or minimize this state of affairs? A good deal of further study is needed. The following measures are suggested as an initial basis for further study:

1. Each pilot before embarking on his career should have an audiogram recorded of his hearing.
2. An attempt should be made to cushion the pilot in an effort to reduce the intensity of his muscolo-skeletal vibrations.
3. The institution of alternate periods of rest from flying duties, with audiometric studies in the interim to determine whether or not such freedom from flying lessens the injury to the auditory mechanism.
4. Microscopic studies of cochleae and their contents when specimens become available.

CONCLUSIONS.

1. The noise and vibrations incidental to flying appear to be injurious to the aviator's inner acoustic apparatus.
2. Audiographic studies and other functional tests unmistakably indicate the presence in these pilots of symptoms pathognomonic of otosclerosis.
3. The amount and extent of injury to the acoustic mechanism of the pilot appears to be directly proportional to his flying time.
4. Aero-otosclerosis as an entity has arrived. Its prevention in the pilot should be a concern of the otologist.
5. Further study is manifestly indicated.

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1433 Medical Dental Building.

**LOS ANGELES SOCIETY OF OPHTHALMOLOGY
AND OTOLARYNGOLOGY.**

The following list of officers in the Los Angeles Society of Ophthalmology and Otolaryngology for 1938 were appointed: President: Dr. Clifford B. Walker; Vice-President, Dr. Leland G. Hunnicutt; Secretary-Treasurer, Dr. John P. Lordan; Committeeman: Dr. Sylvan S. Goldberg.

Place: Los Angeles County Medical Association building, 1925 Wilshire boulevard, Los Angeles. Time: 6 p.m., fourth Monday of each month from September to May, inclusive.

THE TRUTH ABOUT THE X-RAY TREATMENT OF DEAFNESS.

DR. HAROLD HAYS, New York.

In a recent article by R. Sann (*Hals-Nasen-Und Ohrenarzt*, July, 1937), on the effect of X-ray irradiation of the stellate ganglion in cases of deafness, we are given to understand that 23 cases were treated and that 19 cases showed definite improvement. Perhaps Dr. Sann treated selective cases because he states that all these patients showed sympathetic nervous system disturbances.

My main object in bringing out the truth is to prevent the X-ray specialist from immediately beginning to capitalize the statements in this paper; and also to prevent him from treating deafness with "small doses of X-ray" in a manner similar to that advocated by a certain ex-Surgeon-General of the Navy some years ago. If I remember rightly, his idea was to stimulate the pituitary gland and thus cause a reactivation of the entire auditory apparatus. No method could have been worse; no man could have been more self-deluded. Not only was the treatment useless, but most unfortunately this treatment was endorsed or at least tried by a number of reputable roentgenologists who promised much.

Primarily, let us start out with the basic fact that X-ray treatment cannot cure deafness any more than such treatment can cure diseased tonsils. If we can come to an agreement on that point, we shall utilize the services of the X-ray specialist in those cases in which he can actually be of assistance. Once having agreed on the above premises, we are in a position to be fair to the roentgenologist and to the patient.

Like many men who have specialized for a number of years and who have come in intimate contact with hard of hearing patients, I am in a position to analyze these patients so that I can make a differentiation of the type of deafness and advise treatment suitable to the individual case. Having been intimately associated with Leagues for the Hard of Hearing,

particularly through the American Association for the Hard of Hearing, I have seen to what lengths these deafened people will go to obtain relief. I have heard their pathetic stories. Moreover, I have thus been brought in contact with the otologists of this country who are particularly interested in the problem of the deafened. The conclusion of the majority of the members of this national association and of the otologists connected with it is that X-ray treatment of this malady is absolutely useless in the majority of cases; however, one should not go to too opposite an extreme. The X-ray is a valuable adjuvant to treatment in certain selected cases. I have frequently advised this method of treatment in many of my own cases and have urged out-of-town otologists to use it on patients whom they have sent to be for an opinion.

It is timely now to state what I believe X-ray treatments can do and what they cannot do. To place the cart before the horse, one emphatic statement can be made at the present time—this type of treatment is absolutely useless in cases of otosclerosis, nerve deafness or any internal ear condition. However, we have definitely proven its value in those patients who have a conduction deafness in which there are obstructive lesions in the Eustachian tube or nasopharynx, parts that can be reached by X-ray irradiations.

A short time ago, I asked Dr. Herman B. Philips, who has given X-ray treatments to a number of my patients, to send me a list of those he had treated by this method since 1930. There were about 50 cases in all. An analysis indicates that quite a few showed improvement, although there were a number of failures which can be partly explained by the fact that they were patients who were extremely hard of hearing and who were advised to take X-ray treatments in the hope that the pathology in the Eustachian tubes and nasopharynx would clear up sufficiently so that local treatments could be given later on.

In what type of cases was the X-ray treatment of value? Only in those cases was it of value in which there was definite pathology in the nasopharynx and Eustachian tubes which could be overcome by irradiation. Our examination of all deafened patients is most painstaking. We are more concerned with discovering basic etiological factors than in mak-

ing lengthy hearing tests. Taking it for granted that the conscientious otologist has noted the pathology in the nose and throat, such as nasal infections, inflammations and obstructions, and the presence or absence of diseased tonsils, we shall confine ourselves to the examination of the nasopharynx and the Eustachian tubes. The postnasal space and nasopharynx can be examined satisfactorily only with a nasopharyngoscope. In certain cases of deafness, one notes an increase of lymphatic tissue in this region, particularly the parts adjacent to the Eustachian tubes and the fossa of Rosenmuller. In other cases, he will note a turgescence of the mucosa, an edema or even a polypoid condition of the mucosa. In such cases X-ray therapy should be advised before any attempt is made to give local treatments.

After the examination of the nasopharynx, one must determine the pathology within the Eustachian tubes. Obstruction may be due to many factors; in a certain percentage of cases one may surmise that there is a lymphatic infiltration into the tubes, up to the isthmus and sometimes beyond it which can be overcome in one way only, that is treatment with the X-ray.

After the above examinations have been made, we determine the motility of the membrana tympani. In many cases of deafness the drum is freely movable, indicating that there are no adhesions in the middle ear. In rare instances (usually when the Eustachian tubes are wide open) there is a relaxation of the drum which is difficult to overcome.

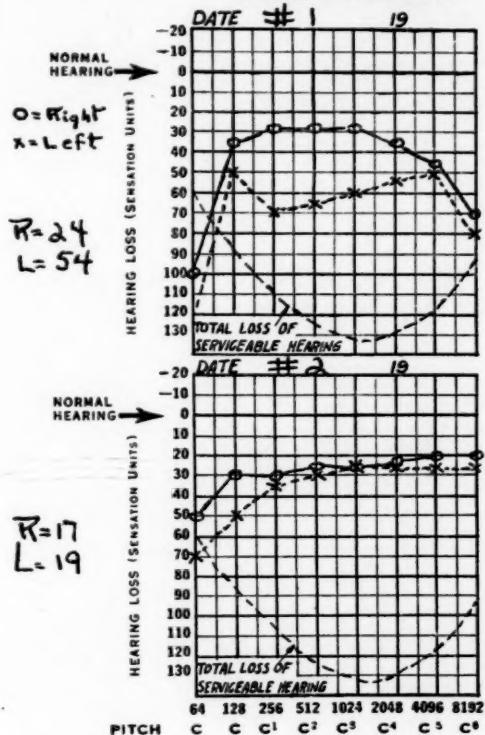
Dr. Philips uses two types of treatment. First, the rapid type for out-of-town patients. A treatment is given every other day, first on one side and then the other, until eight treatments in all have been given. Second, the slow type of treatment which is to be preferred—an irradiation every two weeks for ten treatments.

No medication of the nose, throat or ear is allowed during the course of treatment.

In selected cases we have found that X-ray irradiation is of inestimable value in about 30 per cent of the patients with nasopharyngeal pathology. A few of these cases have been so improved that it has been unnecessary for us to pro-

ceed with further local treatments. Others have been followed up and treated locally for an indefinite length of time. Of primary interest is the discovery that the nasopharynx and Eustachian tubes have changed their appearance. In some cases no lymphatic infiltration or edema can be seen after the course of irradiation.

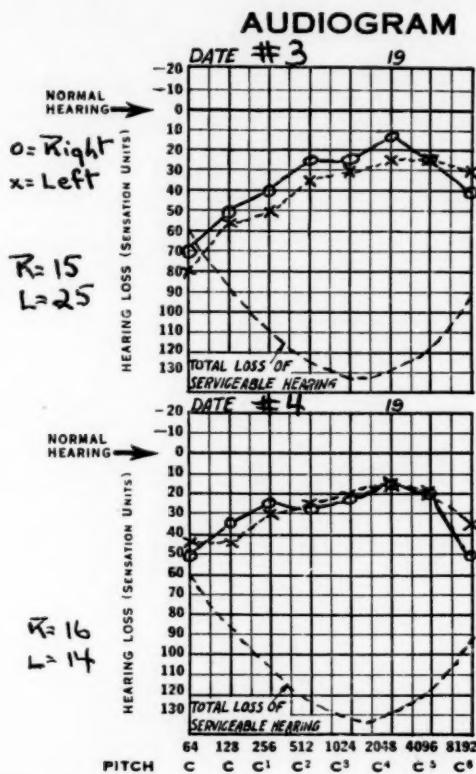
AUDIOGRAM



A citation of two cases should prove of interest. I shall outline them briefly:

Case 1: A young school teacher who had had a number of operations on her nose so that there was a tendency towards an atrophic condition of the mucosa, presented herself a few years ago. Examination with the nasopharyngoscope showed

polypoid masses extending into the nasopharynx and a hypertrophy of the mucosa of the Eustachian tubes so that there was almost a complete stenosis. Local treatment was tried for a number of months with little improvement. She was finally prevailed upon to have X-ray treatment. A test of



her hearing with the audiometer some months later showed marked improvement (see audiograms 1 and 2).

Case 2: A young married woman, much concerned about her deafness, was examined about eight months ago. Local treatment had been tried by other otologists. Her hearing became worse. Examination with the nasopharyngoscope

showed a lymphatic infiltration of the nasopharynx and it was felt that the same pathology was present in the Eustachian tubes. Both tubes were almost completely occluded. She was referred to Dr. Philips, who gave her ten X-ray treatments during the summer months. On her return to our offices we were happy to find that her audiogram showed normal hearing (see audiograms 3 and 4). What is of more importance is that the patient feels that she has normal hearing.

In conclusion, I wish to make the following statements:

1. X-ray treatment for deafness should only be attempted in selected cases.
2. In the majority of cases of deafness this type of treatment is useless.
3. In those cases in which there is a definite pathology in the nasopharynx (as determined by a minute examination with the nasopharyngoscope) and in the Eustachian tubes, proper X-ray irradiation may in itself improve the hearing or may clear up the pathology sufficiently to allow of suitable local treatment.

Note: Many otologists have requested instruction about X-ray treatment. The following is sent to them:

X-RAY TREATMENT TO NASOPHARYNX AND EARS IN CASES OF DEAFNESS OUTLINED BY DR. HERMAN B. PHILIPS.

Two types of treatment have been given, the first in which it is desired to give them in as short a time as possible, the second where there is no hurry on the part of the patient. The first is associated with slight reaction and irritation of the throat. The second is not accompanied by any symptoms or irritation, whatsoever.

In the first, treatments are given over the side of the neck, including the ear back as far as the hair line and inward to the angle of the jaw to expose the entire nasopharynx, larynx and ear. Treatments are given every other day, four (4) on each side, totaling 80 per cent of a skin erythema dose, using the following factors: 180 kilovolts, 4 milliamperes, $\frac{1}{2}$ mm. copper and 1 mm. aluminum filtration; 60 cms. distance.

The usual precautions against using irritating chemicals to the nasopharynx and throat for at least six weeks following the treatments and heat or irritating chemicals on the skin about the ears and side of the neck must be followed rigorously.

The slower treatment is given with a smaller machine, using the following factors: seven minutes to each side of neck and ear, 15 inches distance, 4 mms. aluminum filter, 5 milliamperes, 9-inch gap.

The treatments are given every two (2) weeks for about ten (10) treatments.

The precautions against intensifying the X-ray effects by heat and chemical irritation are the same.

136 East 57th Street.

CHICAGO TUMOR INSTITUTE.

The Chicago Tumor Institute opens March 21, 1938. It will offer consultation service to physicians in the diagnosis and treatment of cancer and radiation facilities for cancer patients.

The Institute also proposes to conduct research and to offer training to physicians who may wish to qualify as specialists in the study and treatment of this disease. Dr. Max Cutler, Director, 21 West Elm street, Chicago.

AUDIOMETRIC STUDIES ON SCHOOL CHILDREN.
THE USE OF AUDITORY MASKING
IN HEARING TESTS.*

DR. ANTONIO CIOCCO, Baltimore, Md.

INTRODUCTION.

It is the consensus of opinion among otologists¹ that in making accurate measurements of hearing acuity, a masking device should be employed to eliminate the participation of one ear when testing the other. Masking is especially advised whenever bone conduction acuity is being measured and in those cases in which the thresholds for air conduction differ in the two ears. The theoretical objective of masking is to eliminate perception of sound in one ear (the masked ear) without affecting the hearing acuity of the other ear (the tested ear).

While the necessity for masking is generally recognized and the objective well defined, until now insufficient consideration has been given to the technique of masking. In particular, little attention has been paid to the fact that the theoretical objective of masking is not fully realized in practice. The introduction of a masking sound in one ear interferes with the hearing acuity of the tested ear, but the degree of this interference has never been measured. Moreover, no adequate criterion has yet been proposed to reveal when elimination of perception in the masked ear has been accomplished. Consequently, the application of masking as a routine audiometric procedure has at present limited utility, inasmuch as an explicit interpretation of the results of masking cannot be given. If the masking intensity is made sufficiently loud so as to give visible evidence of having blocked perception in the masked ear, it not only produces a disagreeable physical sensation in the subject being examined, but has apparently a marked effect on the perception of the tested ear. If, instead,

*From the Division of Public Health Methods, National Institute of Health, United States Public Health Service, Washington, D. C., and the Department of Biology of the Johns Hopkins School of Hygiene and Public Health, Baltimore, Md.

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a weak masking intensity is used, one is uncertain whether the main purpose of masking has been achieved. In either case, the effect of the masking sound on the hearing acuity of the tested ear being uncertain, the results of the test will have doubtful value as far as determining the significance of hearing impairment at the time of the test or in comparison with previous or subsequent tests.

It is apparent, therefore, that if audiometry is to benefit from the routine application of masking, it is necessary to arrive at some standard procedure of masking, which will permit comparability of results. An attempt to develop a technique for the determination of the minimum masking intensity sufficient to eliminate perception in the masked ear is reported here, together with the results of its application in the examination of the hearing of 300 children.

MATERIAL.

During the scholastic year 1936-1937, a longitudinal study initiated in 1930 by the United States Public Health Service on the hearing of a sample of Washington, D. C., school children was continued. In 1930 the hearing of a group of 1,400 school children was measured for both air and bone conduction acuity with a 2-A audiometer.² Otoscopic and nose and throat examinations were also included. About three years later it was possible to re-examine 543 of these children in the same manner.³ During 1936-1937, an attempt was made to examine as many of the original group of children as were still enrolled in the Washington schools. Altogether 577 children were examined, the majority of them for the third time. There were 288 boys and 289 girls, their ages ranging from 11 to 19 years, with a mean of 15 years. As in the previous examinations, these children were given an otoscopic and a nose and throat examination;* the hearing acuity for both air and bone conduction was tested with a Western Electric 2-A audiometer. In contrast with the procedure used in the first two surveys, the hearing tests made in 1936-1937 were conducted in sound insulated booths and included tests with a 512 d.v. tuning fork. Tests with the tuning fork were limited, however, to the determination of lateralization of the

*The otoscopic and nose and throat examinations were made by Meyer M. Baylus, M.D.; Edward M. O'Brien, M.D., and Sander E. Lachman, M.D. The hearing tests were made by Mrs. Minerva M. Masinecup and Miss Delta Moore.

fork from the midline (Weber test) and the measurement of relative length of perception by air and bone conduction (Rinné test). A Witting-Pilling auditory masker[†] was used, also, in connection with the bone and air conduction measurements of 397 children. Of these, 39 gave unreliable or inconsistent responses for all the hearing tests. In the opinion of the examiners, these children could not or would not cooperate sufficiently in the testing procedures. Since there is doubt regarding the accuracy of the results, the records of these 39 children will not be considered further in this report.

A TECHNIQUE FOR DETERMINING MINIMUM MASKING INTENSITY.

Any attempt to determine the intensity of the masking sound just sufficient to eliminate sound perception in the masked ear must overcome the difficulty inherent in the process of simultaneously masking and testing the same ear. With the masked receiver in operation, no other air conducted sound can be perceived in the ear being masked. Consequently, the technique to be employed must be limited to a means of determining the necessary masking intensity to eliminate perception of sound transmitted by bone conduction to the masked ear. For purposes of the present work it is assumed that elimination has occurred when the masked ear no longer perceives the bone conducted sound of a tuning fork or audiometric receiver—in this instance, a tuning fork of 512 d.v. The minimum intensity of the masker necessary to cause this elimination of perception will be considered the minimum masking intensity to use in testing the opposite ear.

It is necessary to consider also where, on the head, the sound source should be placed. Both the homolateral mastoid of the masked ear and the midline of the head may be chosen; there are advantages and disadvantages attached to both positions. In this first attempt to measure the minimum masking intensity, preference has been given to the latter position.

In brief, according to the technique here proposed and adopted, the minimum masking intensity sufficient to eliminate the perception of the masked ear is defined as the minimum intensity that is necessary to employ in order that a

[†]The masking tone consists of 120 cycles and all multiples of 120 cycles beyond the forty-second. The intensities of the individual components of the tone are essentially equal. E. G. Witting. Personal communication to the author.

vibrating tuning fork placed in the midline of the head is no longer heard in the masked ear.

It is immediately seen from the above considerations that the method here proposed utilizes the results of the Weber test. These are that a vibrating tuning fork placed in the midline of the skull will be heard:

1. Equally well in both ears (not lateralized) if the two ears have the same bone conduction acuity.
2. In the poorer hearing ear, if the auditory defect in that ear is due to an obstructive lesion.
3. In the better hearing ear, if the auditory defect is due to a nerve or inner ear lesion.

Occasionally, the fork is heard in neither ear and then, unreliable responses being excepted, it is assumed that there is a bilateral auditory lesion of the nerve or inner ear type.

Of these four responses, the first occurs in the great majority of the population, the others being typically associated with pathologic conditions.

The different types of results of the Weber test need to be discussed separately in connection with the determination of the minimum masking intensity. The proposed technique for the determination of the minimum masking intensity and the masking intensities used when the method is not applicable can best be understood by a complete description of the method of examination that has been employed.

After the hearing tests for both air and bone conduction were made in the usual fashion, the results of the Weber test were recorded. If the subject after repeated trials asserted that the fork was not definitely heard in one or the other ear, the determination of the minimum masking intensity took place as follows: The intensity dial on the auditory masker was set at 30 decibels, and the masker applied to one ear (the left ear always first). The tuning fork was then struck and held in the midline of the head. The subject was questioned regarding the perception of the sound stimulus. If the fork had been heard in both ears, the dial was set at the next higher intensity (35 decibels) and the process repeated.

The intensity of the masker was increased step by step until the intensity was reached at which the subject reported that he was certain that the fork was heard only in the ear opposite to that in which the masker was applied. The lowest intensity of the masker needed to lateralize hearing completely to the ear not being masked was then defined as the minimum masking intensity to be used in the examination of the ear opposite to that in which the masker had been applied. It should be mentioned that the masker was held to the ear only a few seconds at a time and that while the subject was questioned and the dial changed, the masker was silent. This precaution was considered necessary in order to inhibit or prevent auditory fatigue.

This procedure was not always applicable when, in response to the Weber test, the subject definitely lateralized the fork to one or the other ear. In this event the possible findings are: 1. There is no difference in hearing acuity by air conduction between the two ears;* 2. there is a difference and the fork is lateralized, *a.* to the better hearing ear, *b.* to the poorer hearing ear.

Depending on which condition existed, the following conventions were adopted in this survey:

1. No difference in air conduction acuity between the two ears, the Weber test lateralized to one ear.

In this case the procedure outlined above was employed to determine the minimum masking intensity to apply when testing the ear to which the fork was not lateralized. The same value of the minimum masking intensity was used to test the opposite ear.

2. (a) The Weber test lateralized to the better hearing ear. In this case the same procedure outlined above was employed to determine the minimum masking intensity to apply when testing the poorer hearing ear. The masking intensity introduced in this ear when testing the better ear was established at 30 dcb. higher than the air conduction threshold of the better ear for 256, 512, and 1,024 d.v. In no instance, however, was a masking intensity greater than 85 dcb. used.

2. (b) The Weber test lateralized to the poorer hearing ear. In this case the masking intensity introduced in the better ear was 30 dcb. higher than its threshold for 256, 512, and 1,024 d.v., but never exceeded 85 dcb. In testing the better ear the masking intensity to be introduced in the poorer ear was obtained in the manner described above.

*In this study this means that for the auditory frequencies of 256, 512, and 1,024 d.v., the hearing acuity in the two ears do not differ by more than 10 dcb.

[†]There should be no necessity of applying a masker to the poorer ear when testing the better hearing one. It may, however, be done in order to render all the records comparable, because even under the best of conditions the sound which emanates from the masker is not always confined to the receivers.

The analysis reported in this paper is limited to those cases in which the Weber test showed that the fork was heard equally well in both ears, not lateralized, since these cases form the bulk of the observations. In this material 300 children did not lateralize the Weber test and gave consistent responses to the technique used. They constitute 84 per cent of the children who were examined with an auditory masker and who gave, also, reliable and consistent responses to the complete hearing test.

TABLE I.

Variation in Minimum Masking Intensity as Observed on 300 Washington, D. C., School Children, Right and Left Ears Combined.

Minimum Masking Intensity (in decb.)	Number of Ears
30 and under	13
35-40	59
45-50	155
55-60	144
65-70	139
75-80	57
85	13
Total	580
Mean	59.66 \pm .37
Standard Deviation	13.07 \pm .26
Median	59.38 \pm .46

VARIATION IN MINIMUM MASKING INTENSITY.

In the group of children who did not lateralize the sound of the 512 d.v. fork at the Weber test, the minimum masking intensity varies considerably from individual to individual. The distribution of all the ears according to the minimum masking intensity, presented in Table I*, appears somewhat asymmetric and skew toward the end of the large values. There is a major peak at 45-50 decb. intensity and slightly smaller frequencies occur at the two following intensity classes. The mean intensity is $59.66 \pm .35$ decb., and the median is $59.38 \pm .44$ decb. The average intensity necessary to lateralize the tuning fork from the midline to the left ear (sound introduced in the right ear) is slightly but insignificantly higher than that necessary to lateralize to the right ear. This slight average difference may be the effect of fatigue

*Failure to record observations properly is responsible for the fact that not all of the 600 ears are included in the tabulations.

since the masking sound was always introduced first in the left ear.

Between the right and left ears of the same individual there is a positive correlation which, measured roughly by the Bravais-Pearson coefficient, has a value of $r = + .48 \pm .03$. Although the differences between the two ears have a range of 90 db., 216 of the 274 children, or 79 per cent, show a difference not greater than 10 db.

TABLE II.
Minimum Masking Intensity Found for Right and Left Ears of 274
Washington, D. C., School Children.

Minimum masking intensity (in db.) sufficient to lateralize to right ear.	Minimum masking intensity (in db.) sufficient to lateralize to left ear.							Total
	30 and under	35-40	45-50	55-60	65-70	75-80	85	
30 and under	1	2	2	—	1	—	—	6
35-40	1	8	21	4	3	1	1	39
45-50	—	4	30	20	11	3	—	68
55-60	2	3	19	27	11	2	1	65
65-70	—	2	8	15	27	9	5	66
75-80	—	1	2	5	10	8	2	28
85	—	—	1	—	—	—	1	2
Total	4	20	83	71	63	23	10	274

Mean masking intensity sufficient to lateralize to right ear = $58.69 \pm .53$
Mean masking intensity sufficient to lateralize to left ear = $60.15 \pm .52$
Correlation between right and left ear, $r = .48 \pm .03$

The existence of a wide range of individual variation, together with the positive correlation between the two ears, emphasizes the need for more precise information regarding the effect of masking. It is evident that the reaction to the sound stimulus of the auditory masker, as measured by the technique described, varies from individual to individual. This variation implies that masking done in a haphazard fashion will diminish the accuracy of the results and interpretations unless the physiologic changes due to masking are known with some precision.

VARIATION IN MINIMUM MASKING INTENSITY AND HEARING ACUITY.

The variation in response to the stimulus given by the auditory masker is not closely associated with the variation in hearing acuity, either in the ear to which the auditory masker is applied or to the opposite ear. This is apparent

TABLE III.
Minimum Masking Intensity and Mean Auditory Thresholds of the Ears to Which Masking Was Applied
(Right and Left Ears Combined).

TABLE IV.
Minimum Masking Intensity and Mean Auditory Thresholds of the Ears Opposite to the One Masked
(Right and Left Ears Combined).

Minimum Masking Intensity (in dch.)	No. of Ears	64	128	256	512	1,024	2,048	4,096	8,192	Auditory Frequencies (in Double Vibrations)		
										Stand. Mean dev.	Stand. Mean dev.	Stand. Mean dev.
Air Conduction												
30 and under	13	3.85	5.20	3.33	4.69	7.08	4.36	10.00	6.48	3.33	7.75	1.25
35-40	59	2.63	7.87	2.54	8.72	7.37	8.25	10.34	6.93	2.20	8.25	.76
45-50	155	2.65	9.75	3.06	11.00	7.00	11.31	10.87	10.15	3.55	11.18	2.71
55-60	144	1.46	7.62	1.74	8.00	6.22	6.93	9.58	6.24	2.40	8.49	2.15
65-70	139	2.34	8.19	2.95	8.89	6.91	8.00	10.65	7.75	3.49	9.80	3.56
75-80	57	3.68	9.54	4.39	10.30	9.04	9.23	11.67	10.10	5.53	12.00	2.72
85	13	5.38	10.10	4.62	10.63	8.85	8.77	16.54	7.94	9.62	8.19	6.15
Total	580	2.47	8.66	2.82	9.43	7.06	8.94	10.63	8.37	3.44	10.00	2.63
Bone Conduction												
30 and under	13											
35-40	59	56.67	5.48	56.67	5.10	57.50	6.32					
45-50	155	54.58	6.39	58.47	7.14	56.53	7.48					
55-60	144	53.74	5.83	57.71	7.14	57.58	7.81					
65-70	139	54.20	5.92	57.62	6.16	58.54	7.87					
75-80	57	53.17	6.32	56.40	7.48	57.12	9.70					
85	13	55.18	5.39	56.14	7.07	57.81	9.85					
Total	580	55.38	6.40	56.15	9.06	55.77	8.94					

from the data given in Tables III and IV. Table III presents the mean air conduction and bone conduction acuity of the ears to which the masker was applied for each class of masking intensity sufficient to lateralize the vibrating tuning fork from the midline of the head to the opposite ear. The mean auditory thresholds for air conduction are somewhat higher for those ears which required strong intensity (75 decibels or more). This increase in the mean threshold, while observed for all the auditory frequencies tested, is not statistically significant. Moreover, there is no consistent lowering of the auditory acuity in relation to increase in minimum masking intensity. The data on bone conduction strengthen the conclusion that the intensity of the masking sound necessary to lateralize a vibrating tuning fork from midline to the opposite ear is to a certain degree independent of the hearing acuity of the ear to which the masker is applied. From the tabulations on bone conduction it is seen that for each tone the mean threshold relative to each class of masking intensity may be regarded as fluctuating according to probability expectations around the total mean auditory threshold for the tone.

The minimum masking intensity, as obtained, is apparently not associated with the hearing acuity of the ear opposite to the one to which the masker is applied. This is seen from Table IV. As for the data presented in Table III, there is no consistent increase or decrease of the mean auditory threshold in relation to increase in minimum masking intensity. For very high values of the minimum masking intensity it appears that the mean hearing threshold is somewhat higher than for the other values, but the number of ears involved is too small to give significance to such deviations.

From the facts presented in these two tables, it seems that the intensity of sound necessary to lateralize the vibrating tuning fork from midline to one ear is to a great degree independent of the auditory acuity of the two ears. Consequently, the practice of choosing the loudness of the masking sound entirely on the basis of the auditory acuity of the masked ear or of the opposite ear cannot be justified.

The relative independence of the variations in minimum masking intensity from the auditory acuity of the two ears

is also clearly shown from the observations on children whose hearing is good in both ears. For such children, it is found that the mean minimum masking intensity and its standard deviation are practically identical to the corresponding constants obtained on all the children.

Another demonstration of the apparent lack of association between hearing acuity and minimum masking intensity is found in Table V, which relates the differences in minimum masking intensity to differences in the hearing acuity between the two ears. In the table the differences in minimum masking intensity between the ears are given a minus sign if the intensity used in the right ear is greater than that used in the left. A plus sign indicates the reverse condition. For the auditory thresholds, a minus sign indicates that the right ear had poorer hearing than the left, and a plus sign that the right ear had the better hearing. From Table V it is apparent that differences in auditory acuity between the two ears are not related to differences in the minimum masking intensity of the two ears. From Table V it will also be noted that the majority of averages of auditory acuity are preceded by a minus sign, which indicates that on the average the right ear has poorer hearing than the left. The significance of this fact is not clear. Since the right ear was always examined first, the better acuity of the left ear might be considered as being due to habituation to the test.

For the auditory frequency of 512 d.v. tested by bone conduction, there is apparent a trend which indicates that a stronger masking stimulus is necessary in one than in the other ear when the bone conduction acuity of the first ear is better than that of the second. For example, if the right ear of an individual has better hearing acuity by bone conduction than the left ear, it requires more sound stimulus to lateralize the fork to the left ear than to the right. This trend is apparent only for 512 d.v., the same tone as the fork used in the Weber test. If it has any significance, one could assume that it means that if a vibrating tuning fork placed in midline has a slight but imperceptible tendency to deviate (is heard slightly better) towards the right, let us say, then it would be necessary to apply more sound stimulus to the right ear in order to make the fork lateralize to the left than it would take for the reverse procedure.

TABLE V.
Differences in Minimum Masking Intensity Between the Two Ears and Mean Difference in Auditory Thresholds.
Mean Difference in Auditory Thresholds Between the Two Ears (Left Ear—Right Ear).

**Based on 101 children.
†Based on 64 children.

EFFECT OF MASKING.

It is of practical importance to determine what effect masking one ear has on the hearing threshold of the other ear and whether changes in the thresholds are related to the intensity of masking. It is well known that masking, as done here by introducing a mixed sound in one ear, always lowers the hearing acuity; that is, raises the threshold of the other ear.

TABLE VI.

Minimum Masking Intensity and Mean Difference Between Masked and Unmasked Auditory Thresholds.

Minimum Masking Intensity (in decb.)	Mean Difference Between Masked and Unmasked Auditory Thresholds									
	Auditory Frequencies (in Double Vibrations)									
	128	256	512	1,024	2,048	Stand.	Stand.	Stand.	Stand.	Stand.
Mean	Stand. dev.	Mean	Stand. dev.	Mean	Stand. dev.	Mean	Stand. dev.	Mean	Stand. dev.	Mean
Air Conduction										
30 and under	4.17	4.93	4.58	5.19	3.75	6.50	4.58	3.80	1.67	7.17
35-40	4.58	6.26	3.56	5.90	2.03	5.46	3.22	7.12	1.78	6.30
45-50	6.00	6.78	5.55	6.36	4.03	5.99	4.84	5.97	2.19	5.27
55-60	6.67	6.69	5.56	6.21	4.86	6.45	5.28	5.49	2.64	6.15
65-70	7.09	6.99	6.33	6.96	6.37	6.70	7.81	5.91	3.85	6.77
75-80	9.65	6.20	7.89	6.69	9.30	6.31	11.32	5.89	8.07	6.47
85	10.77	11.24	9.23	10.89	10.00	6.20	10.38	5.36	8.08	4.18
Bone Conduction										
30 and under		.42	4.77	3.75	4.15	6.25	5.82			
35-40		-1.19	6.60	1.61	6.73	4.75	7.67			
45-50		2.90	6.14	4.71	6.63	6.42	7.82			
55-60		3.51	6.40	6.02	7.38	6.57	8.24			
65-70		4.46	6.28	6.47	7.31	5.94	7.55			
75-80		6.23	6.84	9.04	8.29	9.39	8.99			
85		3.85	6.84	5.77	7.56	6.92	8.21			

This effect is immediately apparent from Table VI, which presents the mean differences between the thresholds of the same ear when the opposite ear is unmasked (open) and when it is masked. The air conduction acuity for five tones and the bone conduction acuity for three tones were examined both with and without masking of the opposite ear. The hearing changes produced in one ear when the opposite is masked may be summarized as follows:

1. On the whole, the threshold is raised slightly more for air conduction than for bone conduction.

2. The lowering of hearing acuity is somewhat more marked for tones below 512 d.v. than for tones above this frequency.

3. For air conduction, in particular, increase in the masking sound intensity applied to one ear produces an increase in the auditory threshold of the opposite ear. The rate of increase in the differences between masked and unmasked thresholds is fairly constant for the tones of 128 and 256 d.v., and becomes accelerated for the tones above the latter frequency.

For bone conduction, the relationship between increased intensity of the masking sound and the difference, masked minus unmasked auditory thresholds is not marked. It appears, rather, that regardless of the masking intensity used, the increase in auditory thresholds remains at about the same level of 4 to 7 deb.

The differences between masked and unmasked auditory thresholds may be considered as due for the most part to the masking introduced in the opposite ear, since the minimum masking intensity was found to be independent of the auditory thresholds of the ear to which the masker was applied and, also, independent of the thresholds of the opposite ear. Full interpretation of these findings, however, must await the results of investigation on the effect of masking in relation to differences in hearing acuity between the two ears, and information on the variation of the minimum masking intensity on repeated examinations of the same individual. It is hoped that information on this point can be presented soon. For the present, it must be sufficient to note that the results of the present study indicate the importance of obtaining definite information regarding the effect of masking.

SUMMARY.

This paper describes a method of determining the minimum sound intensity to be employed in auditory masking. The technique proposed utilizes the results of the Weber fork test.

Application of this method to the examination of the hearing of 300 Washington, D. C., school children gave the following results:

1. The average minimum masking intensity is almost 60 decibels. The intensity required for masking varies considerably from individual to individual, but there is a positive and substantial correlation between the two ears of the same individual.
2. The minimum intensity is not related either to the hearing acuity of the ear to which the masker is applied or to the hearing acuity of the opposite ear.
3. Differences between the two ears, in minimum masking intensity, are not related to differences in auditory acuity between the two ears of the same individual.
4. Masking one ear raises the auditory threshold of the opposite ear. This increase in auditory threshold, which is noted only for hearing by air conduction, is proportionately greater than the increase in masking.

Further observations on the effect of masking will be presented in a later publication.

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1901 East Madison Street.

REVIEW OF A CASE OF OSTEOMYELITIS OF THE
SPHENOID BONE EXTENDING TO THE PETROUS
APEX THROUGH THE MASTOID PROCESS
WITH EXENTERATION OF THE MIDDLE
EAR IN 1900.

DR. MAX HALLE, New York.

The very interesting report in the spring meeting of the New York Otolaryngological Academy meeting concerning the relationship of nasal sinus disease and ear infections leads me to report an unusual case which I recounted in 1902 to the Berlin Medical Society. I think it is best to report the case precisely from the record.

In the year 1900, shortly after I entered a cadaver course with Jansen, my chief at that time, Dr. Friedlander asked me to operate a case of acute mastoiditis with him. Neither he nor I were much practiced in oto-surgery.

I had worked only on the cadaver, and had repeatedly witnessed Dr. Jansen's operations; therefore, I did not feel very comfortable when I saw the patient. He apparently had a severe mastoiditis, with intense pains at the tip of the mastoid. The skin was edematous, and there was profuse discharge from the middle ear, with a temperature of 104°. The striking symptoms were the severe headaches, particularly in the parietal region; no signs of meningitis.

At operation under ether narcosis, there appeared an extensive disease process. At the first stroke of the chisel, pus welled up from the depths; the pneumatic mastoid process was filled with pus to the tip. The tegmen-antri and the sigmoid sinus plate appeared intact. All the cells were carefully removed, cleaned with a burr, as Jansen already at that time advocated, until healthy plate appeared throughout. The disease process improved following operation. The fever fell to 100°; the headache diminished; still the patient continued to complain of headaches, leading thus to only a short period of relief.

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The suppuration remained quite profuse. The headache increased, and there appeared beginning signs of meningitis. I, therefore, proceeded to a second operation, exposing the sinus and the dura of the middle fossa. These regions showed no change. Probatory puncture of the sinus showed free blood flow. I then proceeded to radical operation which also did not reveal any essential change, but then I immediately saw, over the opening of the Eustachian tube, pus appearing anteriorly. With a fine curette I followed this avenue. Apparently there was a considerable coalescence in the petrous bone. Carefully I cleaned out the diseased bone and proceeded slowly into the depths, using the burr and curette. Pus continued to flow. I then proceeded with a fine burr to an extensive enlargement of the operative field, as much as the anatomical conditions permitted, whereby I was very careful to avoid the internal carotid. The disease process, however, clearly showed the path which I had to follow. I did not feel particularly comfortable, since this was my first operative ear case, and it proved to be so very difficult. Slowly I proceeded into the depths of the bone, which was remarkably soft and did not cause great difficulties.

The inspection of the area was not particularly good, and I could discern only a small area of dura up to which I could follow the disease process. I did not expose the carotid. As I carefully cleaned out the diseased area and proceeded into the depths, the curette suddenly entered into a definite cavity. Pus issued from it, and directly there followed from the depths severe bleeding. I naturally was somewhat frightened and thought I had injured the carotid, but it was apparently not arterial bleeding and was easily controlled with iodoform tampon; however, I had to finish the operation.

It was clear that I had followed the course of the petrous bone to the apex, and I had recalled very well several preparations from my collection of bones in which I had studied the development of the cells around the internal auditory meatus where the question would arise what one could do for mastoid suppuration which would progress into that area. Such was apparently the case here. But what was the cavity into which I had proceeded? Was it one produced by a diseased process in the apex, or a large unusual terminal cell? An answer to this question I could not secure.

After the operation the meningeal signs diminished. The temperature fell to 100.5° and the headache lessened. Unfortunately, this favorable course did not last long. All the manifestations became progressively worse. After a change of dressing on the fourth day, profuse discharge reappeared from the depths with only very little bleeding, showing little change in the findings. A repeated lumbar puncture did not improve the patient, and two days later, following a progression of the meningeal symptoms, the patient expired.

I was able to secure an autopsy. It showed severe meningitis, which was less pronounced in the region of the tegmen tympani and antri, and was particularly severe in the region of the sella turcica. The cavity of the mastoid appeared clean throughout. The middle ear was in good condition; the passage way formed to the apex of the petrous bone led into an extensive cavity which proved to be the sphenoid sinus. The mucosa of the sinus was very much swollen, was covered with fibrinous exudate, and the bone of the clivus showed progressive suppurative coalescence.

The findings clearly explained the diseased picture. The patient had, complicating a grippe, a suppurative accessory sinus disease which had involved especially the right sphenoid, thus explaining also the unusually severe vertex headache. Rapidly the process had extended beyond the sphenoid bone, passing over through the petrous apex, leading eventually to a severe middle ear involvement, which then dominated the clinical picture. The meningitis was developed primarily in the neighborhood of the sphenoid. It is not likely that an early diagnosis of the sphenoid disease and extensive operation on that area would have controlled the disease process. It was apparently a case of osteomyelitis spreading rapidly into the depths, and it would have been practically impossible to have removed structures deep enough to prevent the spread of the disease to the meninges.

I, personally, had no occasion to examine the patient rhinologically, since I was only called to the mastoid operation. The autopsy proved I could not have performed a more thorough operation. Now, after 37 years, I could honestly say that I never performed a more difficult operation on the ear. I might also point out that I probably could list this

first case as one of the first cases of petrous apex suppuration ever performed on the living, and that the operation followed the path of the internal carotid into the depths.

In presenting this case to the Berlin Medical Society, I, at that time, emphasized this point, but did not suggest this path as a method of approach to the petrous apex. It is, however, similar in some degree to that described by Lempert in the *Archives of Otolaryngology*, February, 1937. Of course, it was only a relatively unrefined technique as compared to the excellently developed approach described by Ramadier-Lempert, which, in my opinion, would be the method of choice in severe cases, because it is the only one giving a clear view of the diseased area, and a good drainage.

SUMMARY.

A case is described of suppurative disease of the right sphenoid, leading to a coalescence of the bone of the sphenoid extending to the petrous bone, and reaching the middle ear. The most prominent symptom was a severe suppurative mastoiditis which was operated. After unsuccessful mastoidectomy, simple and radical, a deep suppurative avenue was found above the tubal opening, leading into the depths. In 1900, the author followed a suppuration over the internal carotid to the tip of the petrous bone by means of a curette from the middle ear, and burr directly to the sphenoid cavity. The suppurative meningitis was the result of the sphenoid suppuration.

667 Madison Avenue.

LARYNGOFISSURE FOR REMOVAL OF INTRINSIC CANCER OF THE LARYNX.*

DR. ARTHUR PALMER, New York.

While the educational value of motion pictures has long been recognized, their use in medical education has been limited. This limitation has been particularly acute in the field of Oto-Laryngology. Of 271 films approved by the American College of Surgeons, only four concern the larynx, two the ear, and two the sinuses—a total of eight films in the field of Oto-Laryngology.

The film shown was produced by the members of the surgical staff of the New York Hospital in the hope that it might prove useful as a supplement to the classroom lecture or demonstration, and stimulate interest in this field of medical education as applied to the ears, nose and throat. No doubt certain improvements can be accomplished in the production of future films. The model shown was loaned by the Clay-Adams Co.

The operation illustrated is that of laryngofissure for removal of intrinsic epithelioma of the larynx. The technique is similar to that employed by Jackson and others. This type of operation is suitable only in selected cases. It has the great advantages that the patient's larynx is saved, that he has a useful voice without artificial aid, and that he breathes through the normal air passages, whereby air conditioning is preserved.

The patient was a male, age 52 years, with a history of hoarseness since February, 1937. This hoarseness increased gradually until only a whispered voice was present. Pain, coughing and difficulty in swallowing were largely absent. Examination on Sept. 25 showed the presence of a tumor mass in the larynx, situated on the right side, anteriorly, partially covering the right vocal cord and extending below the cord. On Sept. 27, tissue from this tumor mass was removed by direct laryngoscopy. The pathological examination showed the presence of epidermoid cancer.

*Read before the New York Academy of Medicine, Section on Oto-Laryngology, Dec. 15, 1937.

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The patient was admitted to the hospital on Oct. 11 for laryngofissure. The blood count, blood chemistry, urine analysis, X-rays of the chest were essentially normal. X-ray of the larynx showed the normal markings to be disturbed in the region of the ventricle.

On Oct. 14, laryngofissure was performed under avertin and procaine anesthesia. After sterile preparations of skin a midline incision extending from the middle of the hyoid bone to below the level of the cricoid cartilage was made. The underlying subcutaneous tissue and muscles were separated by blunt dissection and the larynx was exposed. A transverse incision was made through the cricothyroid membrane and the interior of the larynx was anesthetized with a solution of 10 per cent cocaine through this opening. Incision with saw was made through the thyroid cartilage, with care to avoid piercing the internal perichondrium. The two portions of the thyroid cartilage were separated by hook retractors and the mucoperichondrium was separated bluntly from the internal surface of the cartilage. A tumor mass was present, involving the right cord and the membrane below it. There seemed to be no extension to the left cord. Surrounding mucous membrane well back toward the right arytenoid cartilage, and for a distance across the midline, was excised with angular scissors. Hemorrhage was controlled. The thyroid cartilage was allowed to fall back into place and the soft tissues above it were united with interrupted chromic gut sutures. The incision was closed with clips, and sterile dressings were applied.

There was little postoperative reaction, the temperature only once rising above 99°. The patient was discharged on the twelfth day, postoperatively, with wound healed except for a small granulating area above the cricothyroid membrane. At the present time this wound is completely healed. There are granulations present internally over the denuded area of cartilage. The final result is undetermined. The patient is able to talk in a loud whisper.

667 Madison Avenue.

LYMPHEPITHELIOMA OF THE MEDIASTINUM— METASTATIC.*

DR. LOUIS KLEINFELD and DR. G. SMITH, New York.

The following case presented some rather interesting points:

The patient, a white male, age 40 years, was admitted to Metropolitan Hospital in November, 1936, complaining of cough, loss of weight and a feeling of tightness in the suprasternal area. He had been in a large cancer hospital a year and a half before. (At the time of admission the reports from the original hospital were not available.) About two months before admission to the Metropolitan Hospital he had had what was thought to be pneumonia with pleurisy. On admission, physical examination disclosed rales over the entire chest, slightly exaggerated breath sounds over the right chest and scars of the neck (probably due to X-ray therapy). Routine laboratory tests were negative.

Chest X-ray revealed peribronchial infiltration. Laryngoscopic examination showed a mass in the upper part of the trachea. Tracheoscopy disclosed granulation tissue in the posterior wall of the trachea, biopsy from which was reported as fibroblastic granulomatous tissue. Two weeks later another tracheoscopy was done by the author and a mass was seen on the posterior wall of the trachea above the bifurcation. Biopsy disclosed lymphepithelioma. After this procedure, the patient complained of substernal pain, increased cough and dysphagia. There was a moderate rise in temperature and foul expectoration was noted. A week after the tracheoscopy, slight tenderness was noted over the left side of the neck and X-ray showed a slight widening of the mediastinum. At first mediastinitis was suspected, but another X-ray, taken after the patient swallowed barium, showed a fistula between the esophagus and trachea, communicating with the mediastinum. Consequently, the impression was that there existed a breaking down of the lymphepithelioma of the mediastinum with consequent fistula formation. At this time a careful

*From the service of Dr. L. E. Hetrick, Metropolitan Hospital.

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search was made for the primary focus. Repeated examinations of the nasopharynx were negative. This patient was extremely tolerant so that a laryngeal mirror could be used to examine the nasopharynx, but absolutely no evidence of malignancy was seen; however, through a Yankauer tubular nasopharyngeal speculum, several biopsies were done at random, from the nasopharynx, and these were later reported as lymphepithelioma. Gastrostomy was done, following which the patient's condition became worse, and he died a week afterward. Following his death, a report was received from the original hospital which revealed that a year and one-half before his present admission he had been examined and a mass found in the nasopharyngeal wall with bilateral cervical nodes. Several biopsies revealed only lymphoid tissue, but the patient was treated as a probable carcinoma of the nasopharynx, with metastases.

Points of interest in this case were: 1. Complete regression of the nasopharyngeal mass (as far as examination went) in spite of which biopsies taken from this area were positive. This illustrates the value of a Yankauer tubular nasopharyngeal speculum for investigating the nasopharynx; 2. the presence of a tracheal tumor which after two biopsies was found to be lymphepithelioma, indicating the need of repeated biopsies until a definite report can be rendered; 3. had the information about the nasopharyngeal condition in the first hospital been available, it would have been wiser to assume the presence of a lymphepithelioma behind the trachea, without taking a biopsy, and then giving the patient deep X-ray therapy promptly.

17 East 84th Street.

MYXOCHONDROMA IN THE NOSE OF A CHILD EIGHT MONTHS OLD. REPORT OF A CASE.*

DR. GERVAIS WARD McAULIFFE, New York.

Myxochondroma, of rare occurrence in any region of the body, is found least frequently in the nose. A brief sketch of its history and a survey of the literature attest to its rarity.

Enchondroma has been accepted as an entity since 1836, when Müller¹ first described it histologically. Progress in classification kept step with advances in microscopical pathology, as evidenced by the work of Fayau,² in 1856. Not until 1857, however, did Virchow³ create the name myxoma, explaining that the synonymous term, mucous tissue, introduced in the previous century, had fallen into disuse. Subsequently,⁴ he divided soft enchondromas into myxomatous enchondromas if the cartilage predominates and cartilaginous myxomas if the mucous tissue is more abundant. Mucous enchondromas and chondromas undergoing mucous degeneration form two other groups. Virchow stressed their teratoid nature. The subject is still controversial, as MacCallum,⁵ in 1936, says: "Ribbert maintains the independence of the myxoma as a distinct tumor, which may be combined with cartilage . . . to form myxochondroma."

In 1868, we find the first description by Trerneuil⁶ of myxomatous enchondroma arising from the ethmoid of a boy, age 11 years. A review of the theses by Fourdrignier,⁷ Wartmann,⁸ Auvray⁹ and Mathieu¹⁰ reveals no account of these neoplasms of the nose. Sicard¹¹ includes in his 29 observations only the report made by Trerneuil.⁶ In 1908, Uffenorde¹² recorded the removal of a chondroma having its origin in the ethmoid of a woman, age 21 years; histologically, it showed myxomatous degeneration. Nine reports¹³⁻²¹ of chondromyxoma and myxochondroma published since 1926 record appearances of the tumors in the cranium, neck, scapula, bronchi, sternum and joints of the arm and leg. New^{22, 23} includes no account of these neoplasms in his summaries of current literature on tumors of the nose and throat.

*Read before the New York Academy of Medicine, Section on Oto-Laryngology, Dec. 15, 1937.

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Two articles of interest have appeared in the recent literature. Handousa,²⁴ in 1935, recorded briefly a case of a tumor removed from the right side of the nose of a young boy who had complained of nasal obstruction for six months. The tumor was adherent to the outer wall of the nose only, but blocked the inner fossa. In the pathological report it is described as resembling a reticuloma; positive diagnosis is not made. Rubaltelli,²⁵ in 1937, removed from the nose of a young adult male a tumor which had caused exophthalmos. Results from the pathological examination showed chondroma with a mucocoele.



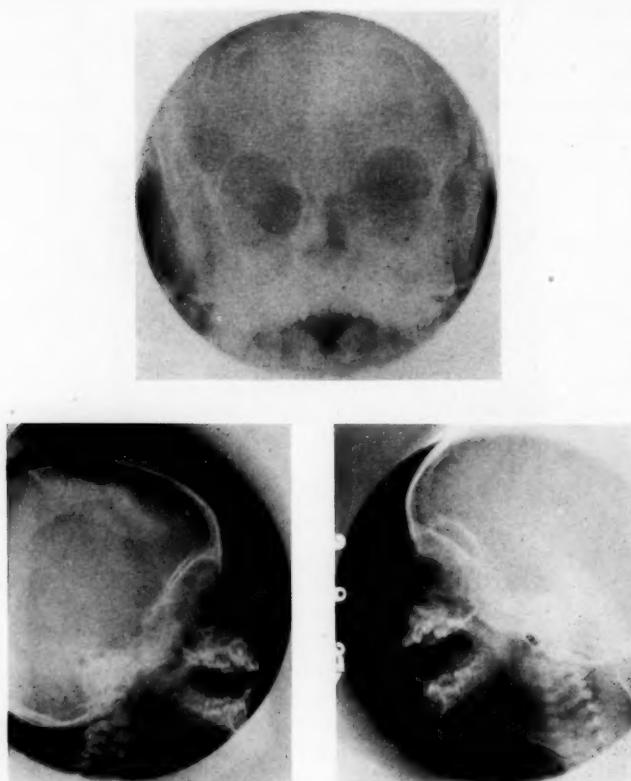
Fig. 1. Myxochondroma in nose of child eight months old.

As far as we have been able to determine, no case similar to the one described here has been presented previously.

REPORT OF A CASE.

A baby boy, age 8 months, was referred to me on Dec. 29, 1936, by Dr. Joyner and Dr. Hunt, of the New York Hospital, because of nasal obstruction resulting from a tumor on the right side. He was a full-term infant, delivered normally; he was delicate, however, and was placed in the respirator for 35 minutes. At first he required one and a half hours for a normal feeding. While he was still in the hospital it was observed that the right eyelid failed to function because of a

secretion. He made satisfactory progress for three weeks, when he developed a fever, and bottle-feeding was begun. Several doctors had advised against consultation with a specialist until the baby was six months old, as displacement of the nasal bone was slight.



External examination showed a lateral deviation of the bridge, with a swelling which seemed to arise from the anterior wall of the maxillary sinus, distending the bridge to twice its normal size. Throat and ears were found to be negative and the baby was referred back to the New York Hospital for X-rays of the skull.

Dr. J. R. Carty, Radiologist to the New York Hospital, made the following report of the X-ray examination on Dec. 30:

"Films of the sinuses and skull show a well defined tumor mass occupying the region of the ethmoid cells and turbinates, more extensive on the right. This tumor has extended upwards anteriorly and downwards, as well as posteriorly. There is some evidence of destruction in the inferior floor and the anatomical outlines are indistinct posteriorly. The outlines laterally on the right are not distinct and may be eroded.

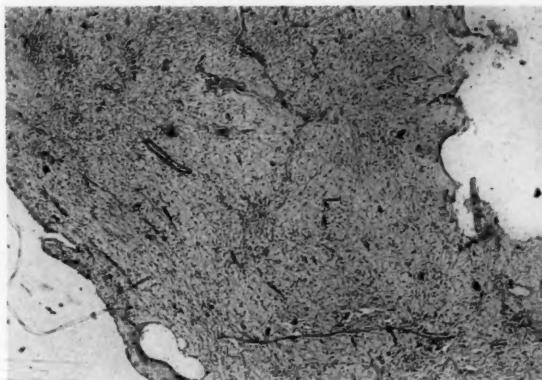


Fig. 5. Low-power view to show the general myxomatous character of the greater part of the tumor, the stellate cells and the cloudy hyaline background of matrix in which they lie.

Impression: Findings suggest a tumor in the nasal cavity which is fairly dense, probably malignant, suggesting an osteochondro sarcoma."

The baby was admitted to the hospital on Jan. 8, 1937, for biopsy. Gross examination of the specimen showed a small piece of tissue 3x2x5/10 mm. in size taken from a tumor of the nose. The specimen was friable, of a dark brown material with a covering of white necrotic-looking tissue. Microscopic examination showed the specimen to consist only of a blood clot, together with a small bit of fibrous connective tissue. There was nothing remarkable about the specimen. Diagnosis was given as fibrous connective tissue and blood clot; no pathological diagnosis was made.

In consultation with Dr. Hocker, it was decided that a second biopsy would be necessary, the tissue obtained by the first having proved insufficient for diagnosis. Accordingly, the baby was readmitted to the hospital on Feb. 10, and a formal biopsy was performed under light anesthesia. A diagnosis of myxochondroma was made, with the following report: "Slides show a mass of connective tissue cells surrounding which is an abundance of mucinous material. Throughout the section there are many groups of cartilage cells arranged in a disorderly fashion. Many islands of normal looking bone can also be seen. There is no evidence of malignant change."

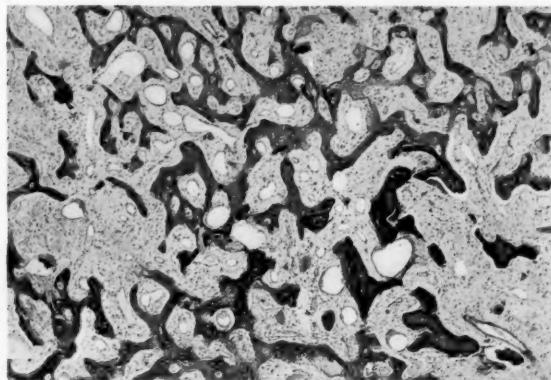


Fig. 6. Osteoid tissue from the tumor. This photomicrograph shows an area in the tumor where the mucoid tissue is rather less predominant, and well marked areas of osteoid and osseous tissue are found. The arrangement of this, however, is comparatively orderly. Osteoclasts are conspicuous by their absence and some of the osteoid tissue shows close similarity to cartilage.

The baby was admitted to the hospital again on March 5, and on the following day the tumor was removed under general anesthesia. An excellent exposure was obtained by a modified Denker operation; the upper lip was incised at the juncture of the gingival margin down to and through the periosteum, and the soft structures of the nose were displaced upwards. The right lateral bony structure was resected sufficiently to dislodge the tumor, which was the size of a small lemon and which sprang apparently just below the attachment of the inferior turbinate. Very slight bleeding was encountered throughout the procedure, and no packing was used postoperatively. The incision was closed with interrupted

mattress sutures. The postoperative course was entirely uneventful, and the baby was discharged on March 10.

The preoperative diagnosis of myxochondroma was verified by the pathological examination of the tumor made by Dr. Foot, who reported:

"Microscopic sections show a tumor that is made up of very irregular, elongated, vacuolated cells with processes. Most of them are fusiform, though a few present stellate characteristics. They lie in a matrix of cloudy, semifibrous mucoid tissues which at times show slight differentiation toward car-

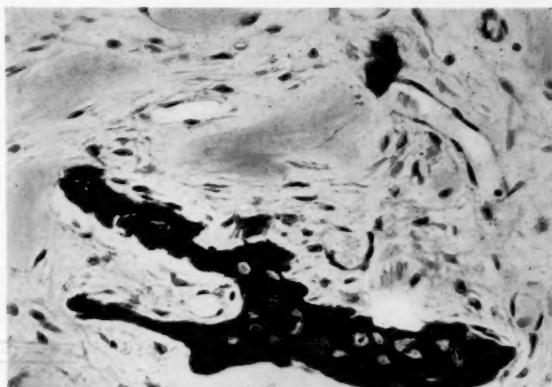


Fig. 7. A spicule of bone in an area similar to that shown in Fig. 6. It will be noted that the osteocytes are not in any way abnormal excepting that they show degeneration. The edges of the bone are "worm-eaten," show no osteoblasts and indicate that the bone is undergoing destruction by the mucoid tumor surrounding it. The myxomatous character of the tumor is well shown in this illustration, and also the good differentiation of the stellate cells, lack of hyperchromasia and lack of mitotic figures.

tilage, particularly around the periphery where there are a few nodules of well-formed, normal hyaline cartilage. Mitotic figures are not noted. Other sections show small bits of bony trabeculae imprisoned in the tumor, apparently having been included. In the neighborhood of the bone there is also a suggestion of osteoid tissue. A third section shows even more bone and more of the osteoid elements.

"Preliminary diagnosis is fully substantiated in this tumor. Academically speaking, it appears to be a somewhat teratoid tumor in the nature of neoplastic development of a fetal rest. While all the elements of osteogenic sarcoma are present, the

cells do not resemble sarcoma cells on account of the good differentiation, lack of mitotic figures and the lack of giant cells. Of course, infiltration cannot be judged from the specimen, but there is no indication that it is an infiltrating growth. Experience leads one to mistrust these "half-baked" cartilaginous tumors, and it would be advisable to keep the case under observation, as recurrence is not unlikely and a malignant change not impossible."

SUMMARY!

A myxochondroma arising from just below the attachment of the inferior turbinate was removed by a modified Denker operation. The case is being kept under observation for possible recurrence and malignant change. A brief review of the literature is given.

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110 West 55th Street.

THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

The 1938 Annual Meeting of the Society will be held at the Claridge Hotel, Atlantic City, Wednesday, Thursday and Friday, April 27, 28 and 29, 1938. Rates are quoted as follows: European plan—single rooms, \$4, \$5, \$6 a day; double rooms, \$6, \$7, \$8 a day. American plan—single rooms, \$8, \$9, \$10 a day; double rooms, \$14, \$15, \$16 a day.

The President, Dr. Samuel J. Kopetzky, has arranged a scientific program of unusual interest and merit. Mr. V. E. Negus, of London, and Prof. Henri Coutard, of Paris, will be guest speakers. Recent developments in our specialty will be presented and will evoke lively discussion.

For your information, the following dates are tabulated, all meetings to be held at Atlantic City:

American Laryngological, Rhinological and Otological Society—April 27, 28, 29.

American Bronchoscopic Society—April 30.

American Laryngological Association—May 2, 3, 4.

American Congress of American Physicians and Surgeons—May 3, 4.

American Otological Society—May 5, 6.

ORBITAL ABSCESS. A CASE REPORT.

DR. LOUIS R. EFFLER, Toledo.

Introduction: The following is a rather unusual case of "orbital abscess" in a lad, age 11 years. One unusual feature is concerned with the extent of the nasal polyposis which one does not expect to encounter so early in life. Other unusual features are concerned with the problem of wound closure and the method of self-irrigation on the patient's part which played such a prominent part in his safe and speedy recovery.

Past History: The duration of the lad's present illness was at first stated to be one week. At that time his mother said he had contracted a severe cold in the head, probably flu. When on examination, however, the right naris was seen to be choked with polyps, the mother remembered an auto injury some five years before in which the lad had been struck over the frontal region. This accident may or may not have had some bearing upon his present trouble, although strangely no emphasis was placed upon any nasal symptoms by either mother or patient.

Physical Examination: When first seen our patient had emphysema of both right lids. This was explained by the fact that the lad confessed to blowing his nose violently and frequently in an effort to clear his right nostril of pus.

In addition, the right eye was distinctly *proptosed* and *deviated to the right*. While not an original complaint, *double vision* was soon volunteered by this young but unusually bright lad. The next most striking symptom was *pain*. The lad was somewhat of a stoic, but his mother volunteered that his nights (and hers) had been sleepless for a week due to a severe headache over his right eye.

As before said, the right nostril was choked with *polyps* and *frank pus*. These we quickly assumed to have a causal connection with his right eye symptoms.

Diagnosis: A three-day observation period was first instituted. During this period nose-blowing was strenuously inter-

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dicted. Ice and heat to the right frontal region were alternated in an effort to reduce the swollen lids. Needless to say, none of these methods produced much change in the external appearance. If anything, the swelling may have become slightly worse during this period.

A tentative diagnosis of "*orbital abscess*" had been made at the beginning. This diagnosis was strengthened after three days' observation. It was concluded that the primary cause of the abscess was a nasal sinusitis, probably a suppurative frontoethmoiditis, right, with the probability of polyp formation, extensive, right. The left sinuses were radiologically and clinically negative.

Operation I: On Oct. 2, 1937, a modified Killian operation was performed on the right side. An extensive incision was necessary through the eyebrow and down to the right ala nasi in order to reflect the swollen tissues.

The periosteum was stripped downward for a considerable distance within the orbit without encountering any pathology. A doubt as to the correctness of the diagnosis was even momentarily entertained; finally, however, after stripping down almost to the equator of the globe, our efforts were rewarded by a sudden gush of thick, creamy, foul-smelling pus. The quantity was estimated at approximately 2 ounces.

Now began the investigation into bone pathology: As suspected, the lachrymal bone was almost completely necrosed and furnished the probable portal of entry into the orbit. In addition, the papyraceous bone bounding the ethmoid labyrinth was also more or less necrosed, though no polyps were seen to invade the orbit.

Most of the anterior third of the inner orbital plate was removed at operation. This was necessary by reason of the extensive involvement as well as the posterior position of the abscess cavity. The frontal sinus, right, was easily identified and inspected. Its size equalled that of an average thimble. It was found full of pus and almost one solid polyp; these were removed. In addition, almost the whole anterior and a great portion of the posterior ethmoid labyrinth were exenterated, because each visible cell contained polyps in addition to those dangling free in the nasal cavity. The right sphenoid was also found full of pus and polyps, and resembled closely the right frontal.

The *diagnosis at operation* thus confirmed the preoperative diagnosis. By no stretch of imagination, however, could such extensive polyposis have been visualized at such an early stage of life and without any previous nasal symptomatology. Such a pansinusitis, acute-chronic, suppurative, right, with extensive polypformation and polypoid degeneration has never been encountered by the author in one so young.

Wound Care: It was deemed inadvisable to close up this wound at operation, even though better-than-average drainage could be obtained through the right nostril by means of a full yard of vaselined gauze packed into the orbital cavity and carried out the right nostril. Instead, it was figured that, after two weeks of open drainage, the wound would be in better shape for final closure, and the physical condition of the patient would also be much improved.

Operation II: On Oct. 15, 1937, the patient was deemed ready for the second stage of his operation, *viz.*, cosmetic closure of the operative wound. While the interval of two weeks, during which time the wound had had daily attention, served to improve the lad's general condition, it did not improve his local condition, but rather made it worse.

True enough there was much less frank pus; but, as if in compensation, the proliferation of recurrent nasal polyps proved extraordinary. As though giving a living illustration to the axiom that "nature abhors a vacuum," these polyps grew so extensively as to fill the open wound completely and overflow the surface, and the nasal view was completely obstructed in consequence.

Technique: It may readily be seen that the purposes of this second operation, therefore, were twofold: *a.* To remove the polyps and prevent their future recurrence; *b.* to close the wound, both for cosmetic purposes and the relief of the patient's double vision.

a. The edges of the wound were extended in both directions in order to give better access to its deeper portions. With a flat instrument inserted into the field by way of the right nostril as a guide, the polyps were removed one by one by grasping with a Weil double-curette. Bleeding was controlled as much as possible with adrenalin drops (1-1000). The more superficial polyps were easily removed. The deeper polyps

naturally offered greater difficulties. In their removal, practically every visible cell of the right ethmoid labyrinth that had been overlooked at the first operation was now removed. Every cell was found to have a polyp in situ. The right frontal and the right sphenoid sinuses were carefully inspected and each found again completely filled with recurrent polyps. These also were, of course, carefully removed. More of the bony inner wall of the orbit was found to be necrosed and likewise removed.



The end-result of the operation, thus far, was a yawning cavity that looked as though it never could obliterate itself by granulation.

b. The periosteum at the roof of the orbit and over the bridge of the nose was now carefully freed of adhesions to permit the deviated eye to be attracted inward. Incidentally, the skin was undermined at both sides and subcutaneous attachments dissected loose from all points of adhesion.

A row of chromic sutures brought the subcutaneous tissues together and a row of silk sutures was then used to approximate the skin edges. The end-result was a good cosmetic result. The right eye was only slightly deviated. Drainage was secured through the right naris exactly as at the first operation. A pressure-bandage was applied.

Postoperative Care: The aftercare consisted in removal every eight hours of about six inches of vaselined gauze through the right naris till all was removed. None was replaced. Following the total removal of this gauze packing, a soft rubber catheter was inserted into the wound-cavity through the right naris and the orbit and nasal cavities carefully irrigated morning and evening. In this procedure the patient was very helpful. He learned quickly the trick of correct insertion and self-irrigation by hydrostatic pressure.

Two weeks after Operation II, the wound was practically healed. A small breaking down of no consequence took place at its center. Intranasally, there is now not only no visible recurrences of polyps, but the cavity itself appears entirely obliterated, and the erstwhile troublesome diplopia is completely corrected.

SUMMARY.

1. Orbital abscess was proved at operation.
2. The primary cause was a purulent hyperplastic fronto-ethmoiditis with unusually extensive polypformation.
3. Extension into the right orbit was probably by way of the lachrymal bone.
4. The first operation consisted mainly of evacuation of a large postocular abscess-cavity, partial removal of the inner orbital wall, and partial removal of numerous ethmoid cells and polyps on the affected side.
5. The second operation (after a two weeks' interval for open drainage) consisted of a complete ethmoidectomy, complete exenteration of recurrent polyps, curettage of frontal and sphenoid tissues, and closure of the external wound for cosmetic purposes.
6. The right antrum was not surgically disturbed.
7. The deviated right eye was attracted mesially so that, clinically at least, a troublesome diplopia was corrected.
8. A life was saved and future usefulness assured.

222 Michigan Street.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTOLARYNGOLOGY.

Dec. 15, 1937.

(Continued from February issue.)

DISCUSSION.

DR. CAESAR HIRSCH: Since Dr. Morrison covered the entire subject so completely, I am afraid that I do not have to add very much. I only would like to point out that as early as 1914, Dr. Arthur Herzog, of New York, used quinine and urea in order to produce a prolonged analgesia in tonsillectomies and adenoidectomies, and in 1923, Dr. Robert Sonnenschein, of Chicago, used an alcoholic novocain solution for the same purpose. In 1909, Yankauer was the first one to describe a method of nerve blocking anesthesia in tonsillectomy which was used by Sonnenschein. Yankauer's method was very much improved in 1926 by Dr. Francis W. Gowen, of Philadelphia. I have used Dr. Gowen's procedure with a slight modification since 1927 in a great number of cases, injecting 2.5 cc. of a 1 per cent novocain adrenalin solution at the lateral palatine foramen, and 5 cc. of the same solution into the retrotonsillar region, puncturing the anterior palatine pillar underneath the inferior tonsillar pole. There is no danger for the nerves and vessels running through the sphenomaxillary fossa if a straight needle is inserted into the anterior tonsillar pillar and advanced parallel to the inferior teeth and not further than to the level of the posterior palatine pillar. Anesthesia was in almost all our cases a satisfactory one, mostly more so than with the usual paratonsillar regional infiltration in which we pierce the mucosa on four, five or even more spots. I never saw the occurrence of Horner's syndrome as was published in 1934 by Dr. Meurman, of Helsingfors.

When I was first approached by the Rare Chemicals, Inc., with the request to use eucupin, I was somewhat reluctant, having worked with eucupin shortly after the war. I started at that time to experiment with eucupin, having read reports of Drs. Morgenroth and Ginsberg, who claimed that they had obtained local anesthesia on the rabbit's cornea with a 20 per cent eucupin solution, lasting as an absolute one for 10 days, being somewhat diminished after 13 days and fading away only after 16 days. Morgenroth and his co-workers found eucupin while searching, not for a local anesthetic, but for chemotherapeutic medications against trypanosomiasis, diphtheria and malaria, and thereby discovered the tremendous local anesthetic power of quinine derivatives. As Dr. Morrison has already stated, eucupin is isoamylhydrocupreine, and I may perhaps be permitted to remind you of another very well known and very powerful local anesthetic, namely nupercain, the relationship of which to quinine, which is methylcupreine, is shown in this table.

In the early 1920's I experimented with a 2 per cent aqueous eucupin solution for topical anesthesia of the tracheobronchial tree and was able to obtain complete anesthesia. However, I was discouraged by the occurrence of a whitish discoloring and turbidity of the very tender bronchial mucosa, obviously due to a slight necrosis of the superficial mucosal layers. Since I learned from the paper of Dr. Takats, of Northwestern University, that eucupin does not produce any sloughing when dissolved in an isotonic saline solution, I understood my previous failure was due to the aqueous solution which I had employed, and I made up my mind to use 0.1 to 0.2 per cent eucupin with 1 per cent procaine in Ringer's solution for nerve blocking anesthesia in tonsillectomy. In about 50 cases we observed the same satisfactory results as Dr. Morrison has reported and never experienced any sloughing in the realm of the operating field. The local anesthesia was as satisfactory as it used to be when only a 1 per cent novocain adrenalin solution was employed. The bleeding during the operation was not more intensive than usual, having added 15 drops of a 1 per mille adrenalin solution to 30 cc. of the anesthetic fluid in order to check the vasodilator action of eucupin. Analgesia lasted in the average for 24 hours after the operation. In one case in which I performed a tonsillectomy in a peritonsillar abscess the analgesia even lasted for 40 hours.

Otogenous Cerebellar Abscess. Author's Abstract. Dr. E. Miles Atkinson.

Pathology: A cerebellar abscess starts in the avascular zone situated in the centre of a folium, and this whether infection of the cerebellum has occurred by a perivascular or a vascular route. Extension from its point of origin takes place: 1. Backwards through the folium originally infected, pushing aside but not invading adjacent folia; and 2. inwards as far as the central mass of white matter, which, however, is seldom involved to any great degree in the suppurative process. The abscess thus remains confined not only to the cortex, but to a single folium of the cortex, and adjacent folia are involved only by the surrounding edema. If the infecting organism is of low virulence and the reaction good, so that an adequate capsule is formed, the extent of this edema is minimal, the silent cerebellar cortex alone is involved, and the central nuclei, on whose invasion depends the production of the characteristic localizing signs, are spared. In this is to be found the explanation of "silent" cerebellar abscess, in which only the general signs of increased intracranial pressure are present, but not the focal signs of a cerebellar lesion. These cases are not uncommon. Indeed, a cerebellar abscess goes more often undiagnosed than does a cerebral.

The Route of Infection: This may be one of three—via the labyrinth, the lateral sinus or the triangle of bone between the two, Trautmann's triangle. The incidence of these differs according to whether the ear disease is acute or chronic.

In acute middle ear suppuration, which accounts for only some 20 per cent of cases, the labyrinth is seldom the portal of entry of infection to the brain, though commonly to the meninges. Only 10 per cent of cerebellar abscesses are labyrinthogenic in acute cases, the lateral sinus and Trautmann's triangle accounting for the remainder in about equal proportion.

In chronic middle ear suppuration, on the other hand, it is held by many authorities, particularly those of the German and Scandinavian schools, that the labyrinth is responsible for 50 per cent of cases. The statistics of Turner and Fraser from Edinburgh, however, suggest that this is much too high, and the British school put the incidence of labyrinthogenic cases at about 20 per cent, with again lateral sinus and Trautmann's triangle dividing the remainder equally between them.

Symptomatology: Abscess, like tumor, in the posterior fossa gives more marked signs of increased intracranial pressure than a supratentorial lesion. This may have localizing significance in cases without focal signs. It is also of importance in connection with the danger of doing a lumbar spinal tap in these cases, lest a medullary paralysis result. Vague meningeal signs are not uncommon. The focal signs are well known, and in uncomplicated cases unmistakable.

Differential Diagnosis: 1. From acute labyrinthitis is the most difficult. Particular attention should be paid to the direction of nystagmus and falling, and the laterality of pointing error. Before a diagnosis can be attained, it may be necessary to extirpate the labyrinth so that the cerebellar picture emerges uncomplicated.

2. Cystic serous meningitis; 3. frontal lobe abscess; 4. tuberculous meningitis; and 5. cerebellar tumor are other conditions which may cause difficulty.

Treatment: There is debate about the best method of approach. In the main otologists favor following the course of infection through the mastoid, neurosurgeons exploration through a clean area. If approach is made through the mastoid, a deliberate obliteration of the lateral sinus, whether thrombosed or not, will greatly facilitate manipulation and improve drainage. This procedure has no harmful effects.

DISCUSSION.

DR. MARVIN F. JONES: I think that Dr. Atkinson's presentation, far from being unorthodox, is decidedly orthodox, with possibly one exception. I think most of us have felt that our abscesses of the cerebellum were usually found to be in layer formation in between the various folia or in this area here (diagram). As Dr. Atkinson presents it, and I think very logically, the infection travels along the blood vessels to the folia, and can travel either along the vessels or by an infectious thrombotic process, I suppose,

in the blood vessel itself. That might explain some of the difficulty that we have encountered when having opened a supposed cerebellar abscess and thought our case was well on the road to recovery, there has been an exacerbation of symptoms and we have wondered why, and gone back in and found other abscesses present. It is possible, by the presentation Dr. Atkinson has made, to explain the multiple abscesses which are so commonly difficult to find in a cerebellar abscess.

For a man to say that he has very great success in the cure of cerebellar abscesses would be going rather far. It may be that by studying the course of the vessels which Dr. Atkinson has described, it will lend a little light on the procedure to follow in order to locate these abscesses. I have found one thing about diagnosing cerebellar lesions, and this is common for the neurologist, the neurological surgeon, the otologist or whoever deals with this particular subject; that is, that every time one gets a little courage to lay down arbitrary rules as to which means which in passing pointing, falling and nystagmus, the autopsy room is very apt to be a great cause for chagrin. I think that each one of the so-called arbitrary rules that I have accepted has been disproved at one time or another by the autopsy procedure.

I congratulate Dr. Atkinson on his very complete presentation and also for adding one thing that may be of very great value to our future treatment in this very discouraging situation.

Dr. JOSEPH E. J. KING: Of the brain abscesses, the three varieties we usually have to deal with, outside of metastatic abscesses, are the temporo-sphenoidal, the frontal and the cerebellar, and in that order of frequency. We insist that the otologist or rhinologist operate upon the source of infection in every instance before we operate for the abscess, unless the patient is in such a serious condition that drainage of the abscess must be done. In most instances we insist upon operation on the frontal sinus or mastoid. We have made the mistake of first exploring the brain, only to discover later that the condition was due to an extradural abscess. Therefore, I think this is most important, and one should never forget that the outside part should be done first. I agree with everything that Dr. Atkinson has said, and he could talk until tomorrow about it and not say all about the subject. I shall say nothing about the symptomatology. I shall say only a few words about mechanical localization and lateralization of cerebellar abscesses, show some slides, and say a few words about how we treat them. In order to prevent confusion with what I have said heretofore about brain abscess, I should like to show some slides of both cerebral and cerebellar abscess, because I believe the treatment of these two types is entirely different and exactly opposite.

Lantern Slide Demonstration: 1. The following reasons made us give up tubal drainage. The pictures are self-explanatory: The end of the tube may be forced into the ventricle, or a secondary pocket may form. The secondary abscess kills as well as the primary one. The tube may become displaced. Autopsy shows it has not even been placed in the tract, but in the brain substance. A tract of hard tissue may form around the tube, very much like the thick lining of pleura in a chronic empyema. The patient may walk around with this, and after slight trauma to the head it may break through into the ventricle causing death.

2. There is the typical position of a temporo-sphenoidal abscess with a well defined wall or capsule. Some may rupture spontaneously through the ear and drain externally.

3. The ventricle is either very much smaller or is completely collapsed. The increase in size of the cerebrum is due to the abscess itself plus the enormous edema about it. The cerebral hemisphere in which the abscess is located is about one-third larger than the opposite hemisphere.

4. This idea was discovered accidentally in 1920, and I have followed it ever since. In all cases of abscess we expect to operate upon them just once and have them get well and not need a second operation. No case operated upon has needed a second operation. We take off the bone and fix the dura to the meninges so as not to spread infection. We then remove the overlying cortex. A portion of the presenting abscess wall is removed. The tendency then is for the floor of the abscess to herniate through the

bony opening within the next few days. The main thing is to keep it from coming out too rapidly and too far. We allow it to advance to the surface, and keep it there by lumbar punctures, salines and positioning. It granulates over and heals.

5. Series of slides showing technique of operation for cerebral abscess step by step. Iodoform gauze is used for packing. Lumbar punctures, position in bed and catharsis, and so on, maintain the brain surface at the level of the skull until the abscess is healed, following which a scalp plastic can be done.

Cerebellar Abscess: There has been great difficulty in lateralizing the lesion. In differential diagnosis I might add to Dr. Atkinson's list the differentiation of abscess from tumor or cyst of the cerebellum.

I think one of the most essential things in the treatment of chronic cerebellar abscess (and that is the one to treat, not the acute abscess) is to reduce the increased intracranial pressure by ventricular puncture. It is most important. If one operates upon them without reducing the intraventricular pressure, there is a great tendency to jam the conus, and if that happens, they die. The other thing that is important is not to allow a therapeutic lumbar puncture. Dr. Browder, of Brooklyn, has made a number of ventriculograms without bad effects. We have made ventriculograms in two cases of cerebellar abscess. Keschner has reported, I think, 24 cases of cerebellar lesion, 21 with homolateral pyramidal tract signs, two contralateral, and one bilateral. As stated, it is important to do a preliminary ventricular tap, and at the same time one can make ventriculograms.

Only two films are needed, the P-A and the A-P. Those I have seen are exactly alike. You find the ventricles dilated and the angles or corners rounded-off or blunted. The third ventricle is dilated. The ventricles on the A-P view show symmetrical dilatation. On the P-A film, one ventricle is smaller than the other. The cerebellar abscess is on the same side as the small ventricle.

I just returned this afternoon from Birmingham, where I attended the meeting of the Southern Surgical Association, and I did not have time to make good slides. Here are some crude drawings made on glass slides with colored pencils.

1. Roughly, this shows the petrous, the foramen magnum, and the cerebellum. A number of cases may have a cortical excoriation of a cortical abscess, as Dr. Atkinson has said, and will be seen on the anterior inferior surface of the cerebellum adjacent to the posterior surface of the petrous. How the infection gets there, I do not know, but it does.

2. Wholly intralobar abscesses. I do not know how many Dr. Atkinson has seen, but in my experience, and from what I can learn from specimens and the literature, there are not so many of them. This slide shows an abscess that is actually intralobar, not fixed to the dura. It is subcortical inside the lobe, just as some are found in the cerebrum. The ones I have seen, and I have not seen so many cerebellar abscesses, have been fixed to the dura over the petrous. Of the three types of abscesses—frontal, temporo-sphenoidal and cerebellar—a great many are fixed at some point to the skull, if you can find it. You may not find it when you operate. They are egg-shaped and the small end is fixed to the point where it started. In the frontal abscess it is directed toward the ethmoid region. In the temporal abscess it is directed downward and outward toward the mastoid. In many cases of cerebellar abscess the smaller pole is fixed to the petrous. There is no sense in herniating it away from the fixed portion. It would probably tear loose and spread infection. Herniation might produce torsion of the brain stem and jamming of the conus.

Should a discharge of pus from a cerebellar abscess be seen at the time of operation for mastoiditis, I think it would be the sensible thing to drain the abscess by means of two small rubber tubes through the mastoid wound. Inasmuch as I do not operate for mastoiditis, I have not seen this condition.

We advise making a vertical incision about $1\frac{1}{4}$ inches long, through a clean area posterior to the lateral sinus with the exposure of the occipital bone about the size of a 25 cent piece. A trephine opening is made with the Hudson bone drill; the dura is incised, and the position of the abscess is determined by the brain cannula. The abscess is usually located in the

outer portion of the cerebellum, and not toward the midline. Therefore, the cannula is directed more external, rather than toward the midline. At a depth of about $\frac{1}{2}$ inch the cannula meets with resistance offered by the blunt nearer pole of the abscess wall. When its position has been determined, an opening in the bone about the size of a 25 cent piece is made, the dura is incised in a circular manner so that an area of the cortex about the size of a five cent piece is exposed. The dura is fixed to the cortex by means of electrocoagulation, and narrow strips of iodoform gauze placed over the dural and bony margins. The overlying cortex is removed by suction down to the abscess wall. The "capsule" appears, and is reddish in color. Most cerebellar abscesses are in children or young people. These patients are usually stuporous and some of them are comatose. The first part of the operation is done under local anesthesia until the abscess wall is exposed. Avertin anesthesia is not advocated in the first stage of the operation due to its possible ill effects on respiration. Should one open and drain the abscess under local anesthesia, the patient is very likely to become alert and restless, and make it difficult to introduce the drains.

Therefore, after the capsule has been exposed, avertin anesthesia is administered, and in four or five minutes the anesthetic has had effect. The presenting portion of the abscess wall can then be removed, the abscess cavity emptied, and two small rubber drainage tubes the size of a Dakin tube, which have previously been prepared, can easily be inserted in the abscess cavity without the patient being in a restless state.

Both tubes should be the same length, and have a small lateral opening near the distal ends. The abscess cavity collapses almost immediately about these tubes so that no actual cavity remains other than the space occupied by the tubes. When the tubes are introduced, the distal ends are carried down to the bottom of the cavity which abuts the posterior surface of the petrous, and both are introduced to the same depth. Narrow strips of iodoform gauze are then placed over the brain substance, sufficient to fill the incision. The tubes are fixed to the scalp by means of two sutures.

After the first two days there is very little discharge through the tubes. They are gently irrigated with a small amount of Dakin solution, in one tube and out the other. The tubes should remain in position for eight or ten days without moving them. This allows complete and firm collapse of the cavity about the tubes, so much so that a small amount of granulation tissue will grow through the lateral eyelets of the tubes. By gentle rotation of the tubes, after removal of the sutures which fix them to the scalp, the tubes should be withdrawn about one-sixteenth of an inch or one-eighth of an inch, and the proximal ends cut flush with the scalp, or just beneath the scalp. Safety pins should not be placed in the tubes for fear of displacing the tubes by shifting of the dressing.

The tubes are shortened about one-eighth inch every two or three days. After 35 or 40 days the abscess cavity will have become completely obliterated and replaced by a fibrous-like stalk. The dimpled area in the skin heals in a few days.

Tubes are used in cerebellar abscesses for the reason that there is no ventricle here which can be perforated by the tube. The distal end of the abscess wall or capsule which in many instances is fixed to the posterior surface of the petrous, should not be herniated, and complete obliteration of the cavity takes place about the tubes due to the shape of the abscess cavity, which is long and ovoid. Secondary pockets do not form.

The two cases operated upon recovered following operation in this manner, and only one operation was required, instead of several.

The last 17 consecutive cases of brain abscess, with exception of one, two of which were cerebellar and two of which ruptured into the ventricles, have recovered, excluding those cases with suppurative leptomeningitis.

DR. E. MILES ATKINSON: I haven't anything more to say except to thank Dr. Marvin Jones and Dr. King very much for their discussion. I was glad to hear Dr. King say that with the method he described he had had easy recoveries in cerebellar abscess. I, too, have found that when they do recover, they recover easily. The published mortality of cerebellar abscess in the hands of otologists has been something like 70 per cent in the literature.

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